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THE OPERABILITY OF CARCINOMA OF THE LUNG*

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SURGICAL intervention in primary carcinoma of the lung has been considered futile in the past with few exceptions. This is chiefly due to the assumption that the condition can be diagnosed only in its advanced stages and that widespread metastases occur early. Now that the technic of lung resection has been sufficiently perfected, and the operative mortality associated with this procedure sufficiently lowered, it becomes of more than mere academic interest to reconsider the subject of carcinoma of the lung from the standpoint of surgical therapy. For example, are we justified in accepting as evidence of inoperability those local consequences of carcinoma of the lung which clinically and roentgenologically attract our attention to the possibility of the condition, namely, atelectasis, bronchiectasis, and pulmonary suppuration?

This present study was based for the most part upon 26 necropsy cases. It was undertaken for three reasons: (1) to evaluate methods of diagnosis, (2) to correlate clinical manifestations suggestive of metastases with necropsy findings, and (3) to estimate the possibilities of surgical intervention.

METHODS OF DIAGNOSIS

It is possible to establish a diagnosis of carcinoma of the lung by employing the following diagnostic aids.

1. Clinical history
2. Physical findings
3. Fluoroscopy

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4. Roentgenography
5. Bronchograpy
6. Bronchoscopy
7. Biopsy or aspiration of tumor
8. Diagnostic pneumothorax
9. Examination of pleural fluid or sputum
10. Thoracoscopy
11. Exploratory thoracotomy

Clinical History.—The symptoms presented by the 26 patients with carcinoma of the lung who came to necropsy have been grouped in the following manner:

1. Local pulmonary symptoms
2. General symptoms
3. Symptoms suggestive of direct extension, metastases or unrelated disease (Table I).

Cough, dyspnea and hemoptysis by themselves were not found to be diagnostic. Occurring for the first time in an individual of the carcinoma age they were considered highly suggestive of carcinoma of the lung, particularly when they were associated with pain. Pain due to carcinoma of

TABLE I

SYMPTOMS UPON ADMISSION TO HOSPITAL (26 NECROPSY CASES)

(Patients Observed at the Medical and Surgical Chest Service, Barnes Hospital)

I. Local pulmonary Symptoms (Grouped According to Frequency)	NO. CASES
Cough	17
Chest pain or discomfort	11
Dyspnea	11
Sputum	10
Hemoptysis or "streaking"	6
"Chest colds"	2
(No chest symptoms 5)	
II. General Symptoms	NO. CASES
Weight loss	9
Weakness	6
Anorexia	3
Night sweats	2
Thirst	1
Fatigue	1
III. Symptoms Suggestive of "Extension," Metastases or Unrelated Disease	NO. CASES
Nervous symptoms*	8
Pain in back or extremities (not related to nerve pathways)	7
Urinary symptoms	3
Vomiting	2
Abdominal pain	2
Abdominal enlargement	1
Engorgement of veins	1
Precordial distress	1
Dysphagia	1

*Including pain of typical distribution in two cases.

the lung was found to be more continuous than that due to any other intrathoracic condition with the possible exception of aneurysm of the aorta. In its intensity and distribution it mimicked the pain of ordinary pleuritis, intercostal neuritis or that due to broncholithiasis. Its persistence and intensity put it almost in a class by itself.

Dyspnea was found to be due to a variety of factors. To name only a few, pleural effusion, obstructive emphysema, increased intramediastinal pressure (superior vena caval syndrome) and alveolar involvement. The significance of symptoms suggestive of "extension," metastases or unrelated disease will be referred to throughout the text.

In the majority of cases the onset of symptoms was insidious. In most instances the patients had enjoyed good health previously. They developed slight symptoms referable to the respiratory tract. Sometimes nothing more than a nonproductive cough, mild enough to escape notice for a long time or merely regarded as evidence of a "cold," was noted. It was only when the symptoms persisted or became progressive that medical aid was sought.

In some cases the onset appeared to be sudden, the clinical picture simulating pneumonia which failed to go on to resolution. The chronicity of the condition then resulted in a clinical picture which suggested such possibilities as tuberculosis, lung abscess, bronchiectasis, an ordinary pleural effusion or empyema.

Duration of Symptoms.—The duration of the symptoms (prior to admission to the hospital and from that time until the death of the patient) is of interest relative to the question of early diagnosis. Nine patients entered the hospital less than six months after the onset of their symptoms, seven gave histories of six to twelve months' duration and in eight the symptoms had lasted for more than one year. Two histories were unsatisfactory because the patients were moribund at the time of admission. Curiously enough, the patients coming within the first six months of their symptoms lived an average of 1.9 months after their admission to the hospital, those having histories of six to twelve months' duration when they entered the hospital lived an average of 4.2 months. Then again, those who had symptoms of one to five years' duration apparently entered the hospital in the terminal stage as they lived less than one month following admission.

These statistics are rather discouraging as they seem to indicate that the patient loses much time in coming to a hospital where a diagnosis can be made. They also show that the disease often runs a fulminating course. Evidence is presented, however, that the disease may run a relatively slow course in some instances, six patients having survived two years or more (two, two, two and one-half, three and one-half, four, and five years).

Physical Findings.—In our cases the recorded physical findings merely called attention to the presence of bronchostenosis, pulmonary consolida-

tion or pleural effusion. Atelectasis occurred as the most frequent consequence of bronchostenosis but the typical findings were frequently modified by the presence of a pleural effusion or mediastinal involvement.

Physical findings indicative of bronchostenosis, atelectasis, pleural effusion, etc., became more than suggestive of carcinoma, however, when they were found to be associated with a Horner's syndrome (ptosis, myosis, anidrosis, enophthalmos), paralysis of a vocal cord or of the diaphragm.

Such nerve involvement accompanying pulmonary involvement should be considered as manifestations of carcinoma of the lung until proven otherwise. Unfortunately their presence is indicative of an advanced stage as they imply that the mediastinum has already been invaded by the tumor. The same may be said of signs of superior vena cava obstruction (dilatation of the superficial veins of the thorax and abdomen, local edema, cyanosis, and prominence of the eyes).

It is well known that fever and other evidences of reaction to infection or toxemia occur in carcinoma of the lung. Because of the variations in the development and course of primary carcinoma of the lung sometimes leading to necrosis or secondary infections, striking differences are seen in the individual temperature curves. In our series (not including terminal fevers) the temperature reached a peak above 38° C. (100.4° F.) in one-half of the cases and in two patients the maximum temperature varied between 39° and 40° C. (102.2° and 104° F.). In three patients the temperature curve was practically normal throughout the entire period of observation.

The pulse curve usually followed the temperature curve and at some time exceeded 100 per minute in 23 of the 26 patients. The respiratory rate also varied greatly from time to time, a maximum rate over 30 per minute being recorded for 11 of the 26 patients.

Laboratory Findings.—Secondary anemia occurred frequently although it was not a constant feature even in the advanced stages of the disease. The hemoglobin readings varied from 51 per cent to 105 per cent and the red blood cells varied in number from 2,500,000 to 6,5000,000 per c.mm. The leucocyte counts fluctuated greatly in different patients and even varied markedly from time to time in the same individual. A white blood cell count over 10,000 was recorded at some stage of the disease by 20 of the 26 patients.

Other laboratory tests such as urinalysis and the Wassermann test employed routinely were of value chiefly in recognizing associated diseases.

Fluoroscopy.—We found fluoroscopic examinations before and after the taking of films to be helpful in the diagnosis for the following reasons: (1) fluoroscopy permitted us to determine whether the shadow present could be dissociated from the hilus, (2) it frequently enabled us to determine the shape, position and mobility of the diaphragm. In certain cases a barium swallow helped not only to locate the diaphragm but also to exclude involvement of the esophagus, (3) it permitted of observations upon respiratory shifts of the mediastinal contents, and, (4) aided in the recognition of small fluid levels, particularly after diagnostic pneumothorax.

Roentgenography.—The films of ten of the patients in this series sug-

gested primary carcinoma of the lung. Metastatic tumor was suggested on three and mediastinal tumor on two occasions. The ordinary roentgen ray film did not suggest primary carcinoma of the lung in seven instances,

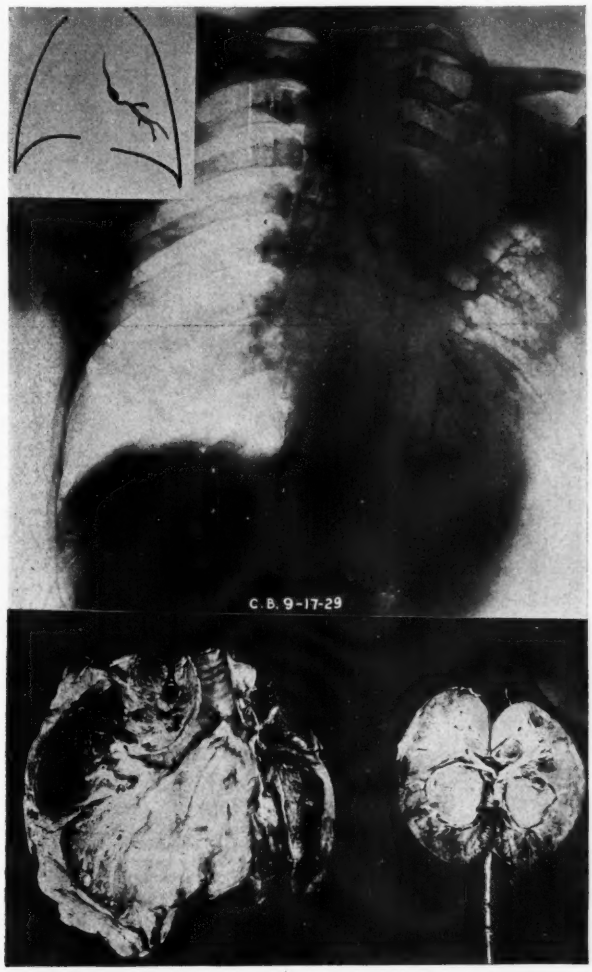

Fig 1.—Primary carcinoma of the lung (left). The lipiodol injection picture shows a filling defect of the left main bronchus. This patient's chief complaint was pain. He was therefore subjected to an alcohol injection of his left phrenic nerve and of the intercostal nerves. This relieved his pain somewhat. Note the appearance of the tumor in this case; also the large solitary metastasis to the kidney. The lesion in the kidney undoubtedly caused some deformity of the renal pelvis but the patient was free from urinary symptoms.

chiefly because of the presence of an associated pleural effusion. The importance of taking more than one film therefore becomes obvious, par-

ticularly after the performance of an aspiration and diagnostic pneumothorax. We need not dwell here upon the various roentgen ray forms of carcinoma of the lung. We have found the classification presented by Lenk of considerable value. This classification includes hilus carcinoma, carcinoma involving a lobe, carcinomatous pneumonia, so-called lymphangitis carcinomatosa, intralobar carcinomatous nodules and the pure intrabronchial tumor. It is of course appreciated that the roentgen ray appearance may be either that of the tumor or of its consequences. In our experience it is chiefly of the latter. We stress the importance of carcinomatous lymphangitis, a diagnostic sign which is frequently overlooked.

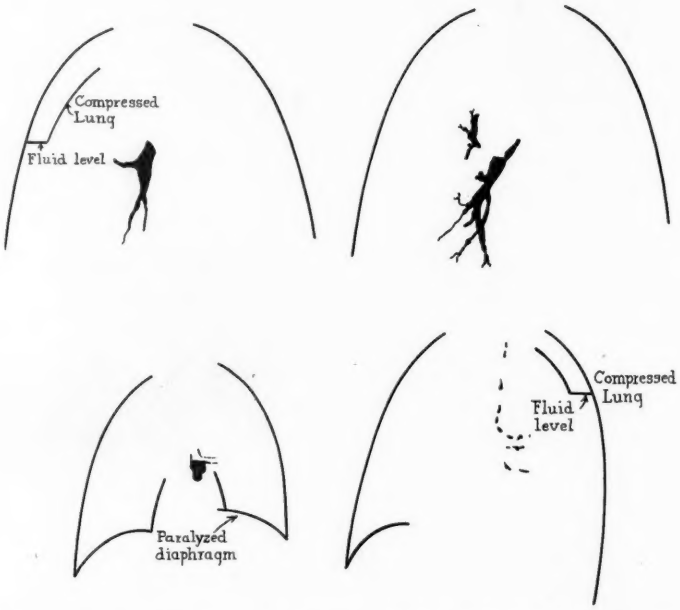

Fig. 2.—These sketches reproduced from films show some of the lipiodol injection pictures which may be obtained in the presence of a primary carcinoma of the lung. None of them may be considered as more than suggestive of carcinoma of the lung for similar findings may be observed in the presence of a bronchostenosis due to a variety of causes.

Bronchography.—Lipiodol injections were performed upon thirteen of the patients. The injection picture showed a filling defect in two and in six a bronchostenosis. External compression of the main bronchus was suggested on two occasions. The method was of no diagnostic value on three occasions. It is our opinion that the demonstration of bronchostenosis or filling defect (Fig. 1) is not diagnostic of carcinoma of the lung per se. We caution against making a diagnosis of carcinoma of the lung on the basis of a lipiodol injection picture alone, for what on one oc-

casion may be considered suggestive of carcinoma may merely be due to an imperfect injection (Fig. 2).

Bronchoscopy.—The value of bronchoscopy in the diagnosis of carcinoma of the lung allows of little discussion. It is the only method we possess of making a positive diagnosis early. (See case H. K., not included in the necropsy series.) Unfortunately only six of the patients to whom we refer received the benefits of bronchoscopic examinations. At the present time, however, such an examination is performed in practically every case.

It is of interest that some abnormality of the tracheobronchial tract was described in five. In one, it was possible to establish the diagnosis by biopsy and to implant radon seeds. In four others the diagnosis was suggested on the basis of indirect endoscopic evidence (stenosis, fixation, deformity of the carina, etc.). In the sixth, dilated blood vessels and evidence of bleeding were the only findings.

Biopsy.—The value of biopsy requires no discussion. The number of occasions on which it proved of value in our cases is noted in the discussion of metastases. It was possible to make a diagnosis in one instance by aspirating the tumor. A large-sized needle was introduced through the intercostal space and the material which was obtained was sectioned in the usual way. We do not recommend this as a routine procedure.

Diagnostic Pneumothorax.—Diagnostic pneumothorax as a rule added little to the data needed for diagnosis, but in one instance after injecting air into the pleural space small nodules having the appearance of metastatic pleural tumors could be visualized on the roentgen ray film. Corresponding metastatic carcinomatous deposits were found at necropsy. In other cases not included in this series Singer has found pneumothorax to be of value in helping to distinguish between pulmonary and extrapulmonary lesions.

Examination of Pleural Fluid and Sputum.—Examination of pleural fluid for cancer cells, according to the Mandelbaum method, was made by Dr. A. Goldman on five occasions. This method of studying the cytology of effusions consists of sedimenting and centrifugalizing the fluid, fixing the sediment in formalin or Zenker's solution, and treating the fixed sediment as an ordinary tissue by running it through alcohols, embedding in paraffin and staining with hematoxylin-eosin. It was diagnostic in three. On two occasions the presence of an empyema complicated the picture. There is therefore some doubt as to whether the pleura was actually invaded by tumor in these two cases. Cancer cells may occur in the sputum, but this is apparently a rare finding.

Thoracoscopy.—Thoracopic examination was not employed in any of our cases. It is recommended by Tudor Edwards and others.

Exploratory Thoracotomy.—Exploration of the pleural cavity is a method of great value not only in establishing a diagnosis but in determining whether operative removal of the tumor is possible and justifiable.

This procedure was carried out on four occasions, confirming the diagnosis in three. In one of these the pleura was found studded with nodules. This method should be reserved for cases in which a diagnosis cannot be made in any other way or in which an attempt to remove the new growth is contemplated. Sauerbruch is of the opinion that exploratory thoracotomy should be performed more frequently than has been the case in the past.

Accuracy of Diagnosis.—Of the 26 patients included in our series a correct diagnosis was made in 17 and in 15 of these the diagnosis was established during the first week in the hospital. In one other patient a diagnosis of primary carcinoma of the lung was suggested but never confirmed. In three patients a diagnosis of metastatic carcinoma of the lung was made. Of the five patients who had diagnoses other than carcinoma, three entered the hospital moribund and died within five days. One did not have the benefit of modern methods of chest diagnosis; in the fifth case the diagnosis was obscured by the development of an empyema.

Of the patients subjected to a thorough diagnostic investigation in the chest service the correct diagnosis was made in nearly all instances.

METASTASES

We investigated the metastases in necropsy cases; first, to determine the frequency with which they occurred in different organs, and, secondly, to attempt to obtain further diagnostic criteria for their clinical recognition.

It is generally assumed that a primary carcinoma of the lung gives rise to widespread metastases early. This assumption is based upon necropsy evidence of distant metastases in the presence of a short history of illness. It is also suggested because of the ease with which tumor cells may invade the tributaries of the pulmonary vein thus entering the systemic circulation directly without the necessity of first passing through the capillaries of the lung.

Five patients in our series were free from symptoms referable to the primary tumor. In four, the symptoms were due entirely to metastatic lesions which happened to be in the brain in three and the spinal cord in one. In the fifth patient the symptoms were produced by an independent disease. In still another, chest pain was the only pulmonary symptom. The chief symptoms in this instance were anorexia, epigastric pain and abdominal enlargement which subsequently proved to be due to metastatic carcinoma of the liver. The experiences of others are similar. Thus Rogers in a recent review of 50 necropsy cases found that in 44 per cent the initial symptom was produced by metastasis rather than by the primary lung tumor. It appears to us that such findings may be used to prove either that metastases occur early or that the primary tumor may remain silent for a long time. This does not necessarily mean that carci-

noma of the lung is characterized by excessively rapid growth and by rapid dissemination. The clinical histories of some of the patients point to a prolonged course which suggests slow tumor growth and late metastases. One of our patients, known to have a primary carcinoma of the lung for 14 months, failed to show any metastases at necropsy while another, save for involvement of one lymph node (removed before death), was free from metastases at the time of necropsy two and one-half years after the appearance of his first symptom. In still a third patient free from metastases the primary carcinoma of the lung was an accidental necropsy finding.

A patient whose history also suggests a prolonged course is still under observation. In this case it might be argued that a chronic inflammatory condition gave rise to metaplastic changes in the epithelium. But is it not surprising that so few cases of carcinoma developing in bronchiectatic dilatations have been described?

C. L. N. (No. 15511) male, aged sixty, was admitted to Barnes Hospital on May 17, 1928, complaining of fatigue and shortness of breath of eight weeks' duration. He had had nonproductive cough for five weeks and chest pain for a few days. He had lost 28 pounds in weight in three months.

His past history made mention of pneumonia at 12, and attacks of "grippe" at the ages of 23 and 50.

Physical examination (1928). The patient was found to be pale and obviously underweight. Examination of his chest revealed diminished breath sounds in the lower half of the right axillary region. There were distant breath sounds and a few moist râles over the left base.

The fluoroscopic examination and roentgen-ray film showed a density in the lower right chest. The lipiodol injection showed a partial bronchostenosis.

A bronchoscopic examination revealed a nodule, apparently malignant, at the junction of the right lower and middle lobe bronchi. A piece of tissue was removed but a diagnosis of malignancy could not be established. The diagnosis of "hyperplasia of epithelium" was reported. The patient was dismissed from the hospital but returned on December 4, 1929. He had continued to be short of breath and on this admission was found to have a sero-purulent pleural effusion. The pus became thick and a thoracostomy for empyema was performed by one of us (H. C. B.). Satisfactory progress was made and the patient was again dismissed. He was readmitted June 24, 1930, because he still had a sinus of the chest wall. This opening was enlarged and a tube was inserted for drainage of the suppurative condition of the lung which was responsible for the sinus. Tissue removed from the sinus proved to be granulation tissue.

The patient again returned on December 14, 1931, because of hemorrhage from the sinus. He would not agree to a bronchoscopic examination. This examination had been indicated for several months because a lipiodol injection revealed a complete bronchostenosis. He later submitted to a bronchoscopic examination which was performed by Dr. Arbuckle on May 21, 1932. The carina was found to be flattened. The wall on each side was elevated into the lumen of the bronchus. The lumen of the left bronchus was obstructed to about one-third, the surface was covered by tissue having the appearance of carcinoma. The right bronchus was obstructed by a pedunculated mass which bled easily. It was obviously a carcinoma which involved the left main bronchus as well. *The tissue removed from both main bronchi showed squamous cell carcinoma.*

Although absolute proof regarding the actual duration of the disease is lacking there is strong evidence that this patient has had a bronchiogenic

carcinoma for four and one-half years and there is no clinical evidence of remote metastases.*

Our findings indicate that carcinoma of the lung is characterized by widespread metastases in its terminal stages in most instances. To repeat, however, there is also evidence that some malignant bronchial tumors

TABLE II

METASTASES IN PRIMARY CARCINOMA OF THE LUNG

(Cases From Chest Service, Barnes Hospital)

M—Metastases found at necropsy. C—Metastases diagnosed (biopsy, tumor cells in pleural fluid) or suggested clinically. X—X-ray evidence of metastases.

NUMBER	INITIALS	CASE NUMBER	AGE	SEX	PLEURA SAME SIDE	LUNG	MEDIASTINUM	REMOTE LYMPH NODES	CENTRAL NERVOUS SYSTEM	SKELETON	LIVER	GENITOURINARY (KIDNEYS)
1	E. B.	M-9498	65	M	M C X	M X	M		C			C
2	C. B.	14058	58	M			M C					C
3	C. B.	21689	42	M				M				M
4	G. B.	20094	45	F	M C	M X				M	M	M
5	J. B.	32527	48	M		X ?		M			M	M
6	D. D.	M-12715	36	M	M C X	X	C	M			M	
7	L. D.	14370	67	F					M C		M	M
8	M. D.	25165	59	F	C X	X ?			M			
9	R. F.	27204	27	M		M	M	M				
10	W. G.	22871	52	M								
11	R. M.	27014	31	M	C X		M X	M				M
12	C. H.	3387	39	M					M C			C
13	A. H.	26526	59	M	M C X	X		M C			M	
14	C. K.	19096	26	F			M				M	
15	J. K.	M-9265	52	M	C				C			C
16	J. M.	31645	54	M	X							C
17	J. Mc.	10143	51	M								M
18	T. M.	16141	45	M	C		M X					C
19	W. Mc.	S-17225	50	M	M C X	M	M	M	C		M	C
20	J. N.	M-1318	36	M		M	M					C
21	F. O.	31860	46	M	C X		M	M C	C		M	
22	F. P.	14604	54	M			M	M				C
23	M. S.	13378	59	F		C X			M C	M X		C
24	C. T.	19396	42	M			M C					C
25	S. T.	9792	75	M							M C	
26	E. D.	33642	45	M					C	M X	M	M C
Totals												
Metastases found at necropsy					5	5	11	9	4	3	10	7
Metastases diagnosed or suggested clinically (including x-ray)					11	7	5	2	8	2	1	12
Agreement of necropsy and clinical findings					5	2	4	2	3	2	1	1

*The patient died at his home on September 13, 1932, after having several pulmonary hemorrhages. No necropsy was performed.

grow slowly and that in the early or even terminal stages no metastases may be present. When one considers the histologic variations which may be demonstrated in carcinoma of the lung it is not surprising that there should be marked differences in the course of the disease in different cases.

We have recorded the metastases to eight important anatomical organs or systems with the corresponding clinical findings which were considered to be suggestive of metastases.

Table II shows that the presence of pleural metastases in this series was always suggested or recognized during life (Table II). The fact that evidence of pleural involvement was more frequently recorded clinically than at necropsy may mean that the pleural reaction leading to effusion is not necessarily proof that the pleura is invaded by carcinoma. Pleural fluid was recognized by the aid of physical findings, the fluoroscope, roentgen ray films and aspiration. Nodules could be observed in the pleura in one instance after a diagnostic pneumothorax. The aspirated pleural fluid was examined for cancer cells by Dr. A. Goldman in five instances and their presence was revealed in three providing complete evidence of pleural involvement.

Regarding pulmonary metastases there was agreement of necropsy and roentgen evidence in only two cases, although lung metastases were found at necropsy five times. This disagreement may be due in part to the development of metastases after the last x-ray film had been taken.

The mediastinum was found to be invaded by cancer on eleven occasions. There was clinical evidence of mediastinal involvement in five patients. One complained of dysphagia during life but no mediastinal involvement was noted in the necropsy examination. Extension or metastasis to the mediastinum may be suggested or diagnosed by precordial distress, dysphagia, marked dyspnea, recurrent nerve paralysis, phrenic nerve paralysis, Horner's syndrome or by evidence of superior vena cava obstruction. Mediastinal involvement may be evident in the roentgen ray film. A barium swallow may aid in detecting involvement of the esophagus.

The ease with which lymph node metastases are recognized depends upon their location. In our necropsy series remote lymph node metastases occurred twelve times in nine patients. Seven of these were in superficial regions, supraclavicular, infraclavicular, axillary or inguinal. Three were diagnosed by biopsy. Of the five occurring in deeper regions, designated mesenteric, retroperitoneal or suprapancreatic, none produced diagnostic symptoms or signs and therefore none were recognized during life.

In the four examples of metastases to the central nervous system reported by us three were diagnosed during life. Table II shows that neurological symptoms or signs occurred more frequently than demonstrable metastases, but this is due in part to the inclusion of vague neurological evidences insufficient for making a diagnosis of organic disease of the central nervous system. It is also true that the brain and spinal cord were not examined in every case. A careful neurologic history and examination

should be one of the necessary steps in the selection of patients suitable for radical surgery. It can also be said that every patient with symptoms of brain or spinal cord tumor should have a thorough study of the chest including an x-ray film, because the neurologic findings may be due to metastases from a silent lung cancer. It is perhaps well to point out that neurologic findings in the presence of carcinoma of the lung need not necessarily be due to metastatic carcinoma. In one of our patients the neurologic symptoms were manifestations of syphilis of the cerebrospinal meninges.

Of the three recorded instances of skeletal metastases two were diagnosed clinically. This probably does not include all such metastases since most necropsies are incomplete in this respect. Careful examinations of the skeleton for areas of tenderness will more often lead to their recognition by subsequent roentgen ray examination. In this connection the case reports by Hirsch and Ryerson are of interest. They found that four patients believed to have endothelioma of bone were actually suffering from bronchiogenic carcinoma with skeletal metastases.

Metastases to the liver may sometimes be recognized by palpation as in one case in our series. In the 33 cases of metastases to the liver reported by Maxwell and Nicholson 9 were recognized clinically. Hepatography with thorotrast may eventually prove to be a valuable aid in recognizing secondary growths in the liver. This method was not used in the series reported here.

In this study we were struck with the frequency of metastases to the kidney (7 of 26 necropsy cases). Although it is usually assumed that kidney metastases cannot be recognized during life we wish to correlate the urinary symptoms and laboratory findings with the necropsy appearances of the kidneys. A glance at Table II shows that urinary symptoms were noted in our series more frequently in patients without kidney metastases. Urinalyses also were more frequently misleading than of value; such abnormalities as albumin, casts or pus cells were due to conditions not related to involvement of the kidney by tumor. The presence of hematuria, however, is more significant. In one of our cases (26 E. D.) a few red blood cells were noted in the urine and as the necropsy revealed kidney metastases encroaching on the renal pelvis the red blood cells in the urine may be looked upon as findings of diagnostic value in this case. Fortunately this patient had been cystoscoped and pyelogram studies had been made a few weeks prior to his death (Fig. 3) but these studies gave no information of positive value in detecting the tumor which must have been present at that time. These metastatic deposits invaded the renal pelvis but slightly. Another example is afforded by Case 3 (see Fig. 1) in which a large mass deformed the renal pelvis. There was nothing in the clinical history or examination of the urine to lead one to suspect kidney involvement. Pyelograms were therefore not made but it is probable that in this case the presence of tumor of the kidney could have been diagnosed by a pyelogram.

In cases in which tumors are found both in the lungs and kidneys the designation of which is primary and which is metastatic may be extremely difficult or impossible during life and subject to controversy even at postmortem. The gross and microscopic appearance of the tumor under consideration seems to establish it definitely as a primary carcinoma of the lung. On another occasion a patient with a metastatic hypernephroma was subjected to a cautery pneumectomy under a mistaken diagnosis. The correct diagnosis was established by necropsy.

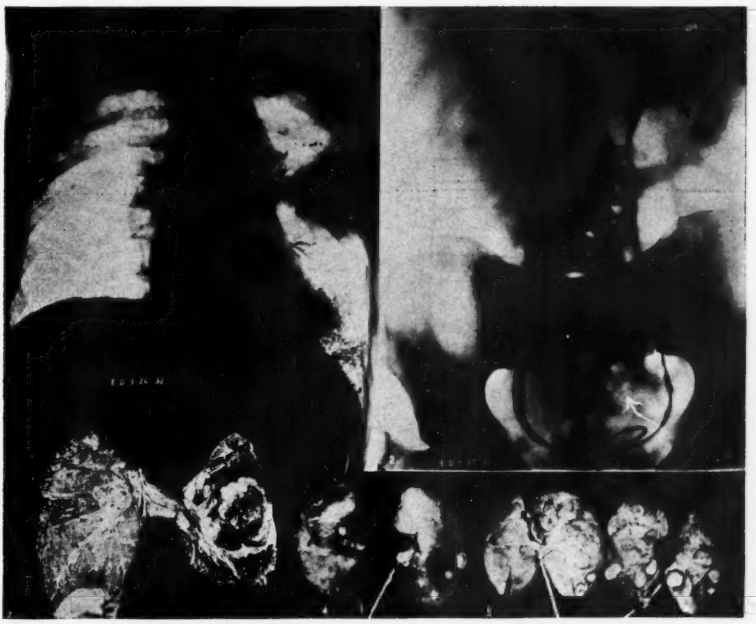

Fig. 3.—Primary carcinoma of the left lung of the intralobar type. Lipiodol in this case suggested a bronchostenosis. This patient complained of pain in the lumbar and sacral regions. He was therefore subjected to a cystoscopic examination. The findings were negative, both pyelograms being normal. The diagnosis in this case had been established preoperatively by aspiration of the tumor. A cautery pneumectomy was considered in this case, but in the absence of pleural adhesions was delayed. The patient, however, developed an empyema which at necropsy was found to be due to an extension from infected tumor tissue. Note the metastases to both kidneys, also the suspicious lesion in the sacrum (indicated by an arrow). The latter was found to be due to an invasion of the sacrum by tumor tissue. The situation of the metastases to the kidneys readily explains the negative pyelogram.

These observations lead us to stress the importance of a complete examination of the genitourinary and respiratory tracts before undertaking a lobectomy for tumor. In this way some primary and metastatic renal tumors will be detected, thus saving the patient the suffering incident to a useless and always serious surgical procedure such as lobectomy.

Other sites of metastases not included in the table (Fig. 4) and their frequency of occurrence are as follows: pericardium (5), heart (1), dia-

phragm (3), chest wall (2), adrenals (8), pancreas (2), spleen (2), peritoneum (2), gastrointestinal tract (1), ovary (1) and skin (1) (Fig. 5). Of the above only the metastasis to the skin was recognized during life. The others remained undiagnosed. The skin metastasis was diagnosed by biopsy. The metastases to the pericardium were not recognized but one of them should have been suspected because the x-ray revealed a pericardial

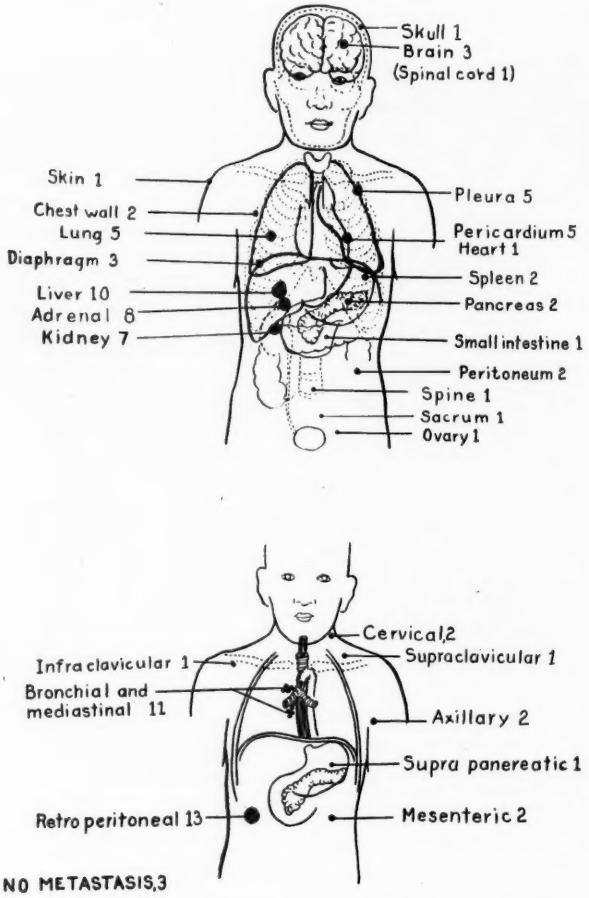

Fig. 4.—The above figures indicate the situation and the frequency with which metastases occurred in the 26 necropsy cases which we report. There were no metastases in three. The above findings correspond in most instances with those recorded by Ask-Upmark (Acta Path. et Microbiol. Scand. **9**: 239, 1932), who reported the findings in 2080 collected cases. They were as follows: Liver, 707, 34 per cent; Skeleton, 405, 19 per cent; Kidneys, 306, 15 per cent; Adrenals, 280, 13 per cent; Central Nervous System (including meninges), 251, 12 per cent; Pancreas, 134, 6 per cent; Thyroid, 83, 4 per cent; Gastrointestinal Tract, 62, 3 per cent; Heart, 60, 2.9 per cent; Spleen, 59, 2.8 per cent; Skin, 32, 1.5 per cent; Ovaries, 24, 1.2 per cent; Muscles, 16, 0.5 per cent; Salivary Glands, 4, 0.2 per cent; Prostate, 3, 0.14 per cent; Tonsils, 2, 0.01 per cent; Testis, 1, 0.05 per cent; and Uterus, 1, 0.05 per cent.

effusion. Involvement of the diaphragm has been found by surgeons who have explored the thorax for malignant disease but it is usually overlooked as it was in our cases. A chest wall tumor may be palpable if of sufficient

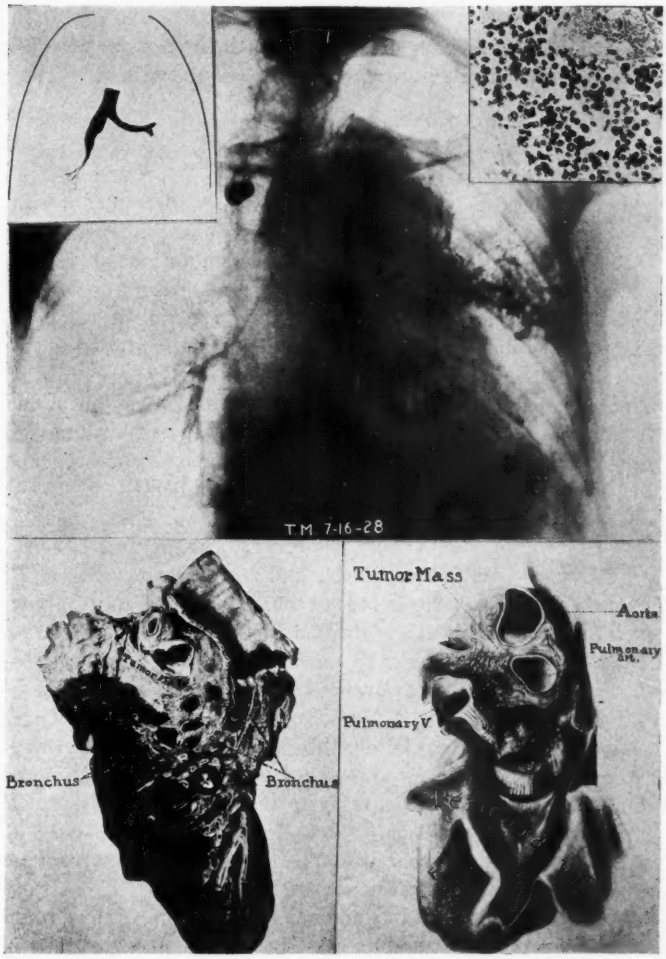

Fig. 5.—Primary carcinoma of the left lung. Note the flattening of the left main bronchus, also the fact that it has been pushed upwards. The normal angle at the bifurcation is no longer preserved because of involvement of the lymph nodes at this point. The dense shadow observed in the roentgen ray film (indicated by the arrow) is a bullet which had nothing to do with the patient's condition. This tumor was of the round cell type and invaded not only the great vessels but also the pericardium and heart.

size or it may cause intercostal neuralgia. In the two examples reported here a diagnosis could not be made. It is apparent that the abdominal viscera may be involved and yet fail to produce symptoms. We were espe-

cially interested to note too that blood pressure readings gave no indication of metastases to the adrenal glands and that metastases to the pancreas occurred in the absence of any evidence of dysinsulinism.

TREATMENT

The treatment of carcinoma of the lung has in the past received consideration along the following lines:

A. Non-operative procedures
   1. Irradiation therapy
B. Operative procedures
   1. Bronchoscopic removal of tumor with or without cauterization, implantation of radium or radon.
   2. Exploratory thoracotomy with implantation of radium or radon.
   3. Extirpation of tumor by lobectomy, partial or complete, by cautery pneumectomy with or without implantation of radium or radon.
C. Treatment directed towards relief of symptoms or complications due to tumor or metastases such as drainage of empyema or lung abscess, relief of atelectasis by partial removal of tumor from bronchus, relief of pain by deep x-ray therapy, injection of nerves, etc.

A. NONOPERATIVE PROCEDURES

X-ray Therapy.—Davidson, Vinson and his associates, Stern, Evans and Leucutia have observed improvement following deep roentgen ray therapy. Evans and Leucutia state that in the early cases repeated applications of deep roentgen therapy and the local use of radium occasionally lead to healing, but in the advanced tumors a temporary reduction in size is all that can be expected. Because of the slow response of primary carcinoma of the lung to radiation, they suggest that the aim should be rather to produce a fibrosis of the involved part of the lung than to act on the carcinoma tissue itself. Contrary to these authors Sauerbruch and Nissen, Maxwell and Nicholson, and Tudor Edwards have been disappointed in the use of x-ray therapy. The latter states that it is doubtful whether even temporary improvement can be obtained.

Our experiences lead us to conclude, for the time being at any rate, that deep x-ray therapy does not prolong the life of a patient with carcinoma of the lung. Further, that carcinoma of the lung is resistant to deep x-ray therapy. The use of deep x-ray in our experience has also failed to relieve pain in such cases. This fact is not surprising, for in the cases which we report the size of the tumor remained unaltered. Relief of pain due to tumor, with few exceptions, cannot be expected unless there is a reduction in the size of the tumor. This failure to respond is not infrequently of diagnostic value, particularly when the diagnosis rests between a pul-

monary and mediastinal tumor. Ten patients not included in the series of 26 cases received x-ray therapy. They are all dead, having lived on an average only five months after treatment was started.

B. OPERATIVE PROCEDURES

Bronchoscopic Removal.—The period of observation is not long enough to justify the claim of a cure in any case except that reported by Jackson.

TABLE III

BRONCHOSCOPIC REMOVAL OF MALIGNANT TUMORS OF BRONCHI

AUTHOR	MICROSCOPIC DIAGNOSIS	DURATION OF SYMPTOMS BEFORE TREATMENT	METHOD OF TREATMENT	RESULT
Jackson, Chevalier: Am. J. Med. Sc. **153**: 371, 1917; J. A. M. A. **89**: 1140, 1927.	Endothelioma	5 years	Tumor extirpated	Alive and well after 11 years
Yankauer, S.: N. Y. Med. J. **115**: 741, 1922.	Inflammatory tissue (?)	2 years	Specimen removed. Radium capsules	Free from cough. Improved (duration ?)
Greene, D. C.: Laryngoscope **34**: 93, 1924.	Malignant tumor, carcinoma, teratoid tumor or hemangioma	2 weeks	Mass removed with forceps. Radium seeds implanted. High voltage x-ray	Followed two months. Cough is only symptom
Ortón, H. B.: Laryngoscope **34**: 97, 1924.	1. Sarcoma (?) 2. Carcinoma	2 years	Pedunculated growth removed	No recurrence in 5 months
Kernan, J. D., and Cracovaner, A. J.: Arch. Surg. **18**: 315, 1929.	Malignant tumor, carcinoma or endothelioma	4 years	Tumor removed piecemeal. Surgical diathermy. Radium seeds implanted	Scar at site of tumor. General improvement at end of 15 months
Arbuckle, M. F.: Unpublished data.	Carcinoma	6 weeks	Removal of tissue. Galvano cautery. High voltage x-ray.	Alive after 4 years. Continues to cough

The patient treated by Arbuckle is still living four years after the bronchoscopic removal of his tumor. The findings in six other collected cases which note the bronchoscopic removal of a malignant tumor are briefly summarized in Table III. There is some doubt as to the classification of the tumor in all cases except that of Arbuckle in which the pathologist gave a report of carcinoma of the lung. The tissue removed by Yankauer was

reported to be inflammatory although clinical evidence favored the diagnosis of malignant tumor. The following case report concerns the patient treated by Dr. Arbuckle.

H. K., male, aged fifty-two, was admitted to Barnes Hospital June 7, 1928. He had had a "cold" six weeks earlier, followed by headaches and afternoon elevations of temperature. There was a productive cough, the sputum sometimes containing blood. There was a sense of constriction in his chest on exertion. There had been a weight loss of fifteen pounds in eight months.

On physical examination there was slight dullness, diminished breathing and coarse râles in right lower chest posteriorly. Fluoroscopy showed a density in the right lower lobe. Lipiodol failed to enter the affected part of the lung. Bronchoscopic examination on June 11, 1928, was discontinued because blood in the bronchi obscured the field of vision.

Bronchoscopic examination was repeated on June 18, 1928. A mass was seen in the right main bronchus having the size and shape of an almond kernel and moving in and out with respiration. It practically filled the lumen of the right main bronchus and had the appearance of carcinoma. A specimen was removed for microscopic examination. The pathologist reported that the tumor had the appearance of a carcinoma arising from bronchial epithelium.

On July 20, 1928, after removal of another specimen the tumor area was treated by the galvanocautery through the bronchoscope. The diagnosis of carcinoma was confirmed by this second biopsy. The galvanocautery was again applied to the involved area on August 3, 1928. The endobronchial treatment was followed by courses of deep x-ray therapy administered in August, 1928, and October, 1929. Since that time he has had no active therapy.

He returned for examination on June 19, 1931, complaining of cough, occasional bloody sputum and occasional elevation of the afternoon temperature. Examination showed that some atelectasis of the right lower lobe still remained.

The patient was last seen by Dr. Arbuckle in June, 1932. His cough was still present and although he was not at work he was nevertheless well enough to take a long pleasure trip by automobile.*

Bronchoscopic removal has been supplemented by other forms of treatment. These include surgical diathermy, galvanocautery, radium in the form of seeds or capsules, and high voltage x-ray. No definite conclusions can be drawn regarding the merit of these different forms of treatment (see Table III).

In one of the patients in our necropsy series radon seeds were inserted bronchoscopically into the tumor by Dr. Arbuckle. This patient was subsequently subjected to a cautery pneumectomy with implantation of radon seeds. He died more than three months later. At necropsy although pulmonary cancer and remote metastases were in evidence no tumor tissue was grossly or microscopically visible at the site of the bronchoscopic implantation of radon seeds (Fig. 6). The local effect of the radon seeds in this case suggests that this form of treatment has possibilities; certainly that it is deserving of further trial, particularly in early cases.

Nelson has shown that with the use of the bronchoscope it is possible to

*Dr. W. H. Field of Evansville, Indiana, informs us that the patient died on September 10, 1932. Necropsy showed carcinoma of lung, septic pneumonia, and metastasis to liver.

insert radon seeds through the trachea or bronchi into the regionary lymph nodes. This procedure is applicable only to the tracheal and interprimary nodes. The plan was tested clinically by Nelson upon two patients and was considered feasible as neither developed mediastinitis.

What Constitutes Operability?—When the diagnosis of carcinoma of the lung without metastasis has been made or the presence of a carcinoma is strongly suggested, then the question of operability should be given consideration in every case.

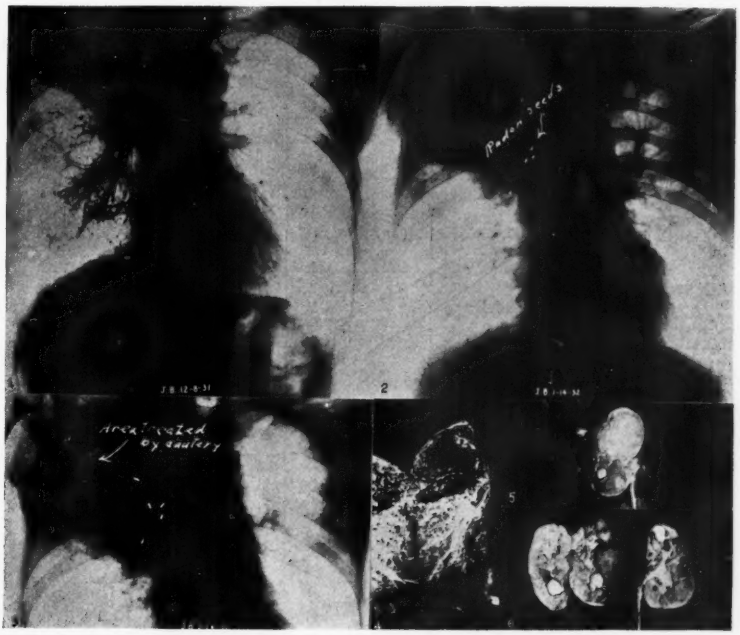

Fig. 6.—Primary carcinoma of the right lung. This patient's complaints were chiefly those of lung suppuration. Lipiodol failed to enter the right upper lobe. A bronchoscopic examination revealed the presence of a tumor. A biopsy was taken and radium seeds were implanted (2). It was later impossible to observe any tumor tissue in the bronchus and so the patient was subjected to a cautery pneumectomy when radium was also implanted. This patient lived for over three months following the latter procedure. At necropsy there was no gross or histologic evidence of tumor tissue at the site of the bronchoscopic implantation of radon, but there were generalized metastases most noticeable in the kidneys.

Thoracotomy will reveal the presence or absence of extension of the growth to adjacent structures. According to Sauerbruch it is the only sure way of estimating operability. Loose attachments to the pericardium, aorta, venae cavae or esophagus are not always considered contraindications to surgical interference. Partial or complete lobectomy should not be attempted in the presence of carcinomatous involvement of the mediastinal lymph nodes. Invasion of a neighboring lobe apparently justifies an

TABLE IV

RADICAL OPERATIONS FOR CARCINOMA OF THE LUNG (COLLECTED CASES)

SURGEON	PUBLICATION	OPERATION	RESULTS	CAUSE OF DEATH
1. Sauerbruch	Chir. der Brustorgane **1**: 849-850, 1920	Resection of lung	Died in five days	Pneumonia
2. Sauerbruch	Ibid.	Lobectomy, left lower	Alive five years	
3. Sauerbruch	Ibid.	Excision of tumor	Died in five days	Pneumonia
4. Sauerbruch	Ibid.	Resection of lung and diaphragm	Lived 3 years, not followed further	
5. Sauerbruch	Zentralbl. f. Chir. **53**: 852, 1926	Lobectomy ?	Successful (follow up ?)	
6. Heidenhain	Quoted by Sauerbruch	Resection for bronchiectasis (carcinoma present)	Died in two months	Other causes
7. Kümmel	Quoted by Sauerbruch and Payr	Pneumectomy	Died in seven days	?
8. Rotter	Quoted by Sauerbruch	Resection	Died in three months	Pneumonia
9. Payr	Arch. f. klin. Chir. **133**: 700, 1924	Resection of lung and chest wall	Died in five months	Recurrence ?
10. Brunn	Arch. Surg. **12**: 406, 1926	Cautery pneumectomy	Seen 5 months after operation	
11. Brunn	Ibid.	Cautery pneumectomy	Died	Pneumothorax
12. Sato	Deutsch. Ztschr. f. Chir. **207**: 424, 1928	Lobectomy	Recurrence in 50 days, reoperation	Died in shock after second operation
13. Hinz	Quoted by Sato and Divis	Left pneumectomy	Died on third day	(?) Died suddenly
14. Walzel	Der Chirurg. **1**: 113, 1928	Resection of lobe and separation from aorta	No evidence of recurrence in 3½ months	
15. Lilienthal	Surg. Clin. N. Amer. **9**: 551, 1929	Lobectomy	Died on ninth day	Bronchopneumonia, collapse of right middle lobe
16. Mirizzi	Sem. Med. **36**: 369, 1929	Lobectomy	Died on sixth day	Fibrinopurulent pleurisy. Fibrinopurulent pericarditis

TABLE IV (CONTINUED)

SURGEON	PUBLICATION	OPERATION	RESULTS	CAUSE OF DEATH
17. Sussi	Gazz. internaz. med. Chir. **39**: 221, 1931	Resection close to hilus	Died on third day	Pneumonia, both lower lobes
18. Harrington	Surg. Gynec. Obst. **52**: 417, 1931	Partial lobectomy	Lived less than one year	Recurrence
19. Harrington	Ibid.	Partial lobectomy	Lived less than one year	Recurrence
20. Harrington	Ibid.	Partial lobectomy ?	Died on seventh day	Cerebral embolism
21. Archibald	Ballon et al. JOUR. THOR. SURG. **1**: 525, 1932	Lobectomy	Died on fourth day	Septicemia
22. Eloesser	JOUR. THOR. SURG. **1**: 485, 1932	Lobectomy	Died in eleven days	Hemorrhage
23. Churchill	15th Annual Meeting Am. Ass'n Thoracic Surg. at Ann Arbor, Mich., 1932	Lobectomy	Died on third day	Bronchopneumonia
24. Churchill	Ibid.	Lobectomy	Recovery (4 months)	
25. Tudor Edwards	Brit. Med. Jour. **1**: 827, 1932	Resection of lung	Free of symptoms two years	
26. Tudor Edwards	Ibid.	Lobectomy (right middle)	Free of symptoms thirteen months	
27. Nissen	Arch. f. klin. Chir. **170**: 118, 1932	Extrapleural type of lobectomy	Operation successful	
28. Allen and Smith	Surg. Gynec. Obst. **55**: 151, 1932	Lobectomy	Alive after two years	
29. Graham	Unpublished data	Cautery pneumectomy, x-ray and radium implants	J. B. No. 32527 Died in three months	Carcinoma of lung with metastases
30. Graham	Unpublished data	Cautery pneumectomy, x-ray and radium pack	C. T. No. 19396 Died in 24 days	Carcinoma of lung with metastases
31. Graham	Unpublished data	Cautery pneumectomy and x-ray	J. M. No. 10143 Died 1st day	Carcinoma of lung with metastases (cerebral embolus
32. Graham	Unpublished data	Cautery pneumectomy and x-ray	A. M. No. 20183 Died in 11 months	? (no necropsy)

TABLE IV. (CONTINUED)

SURGEON	PUBLICATION	OPERATION	RESULTS	CAUSE OF DEATH
33. Graham	Unpublished data	Cautery pneumectomy	C. S. No. 17222 Died in 37 days	? (no necropsy)
34. Graham	Unpublished data	Cautery pneumectomy	J. W. No. 5536 Died in 25 days	? (no necropsy)
35. Graham	Unpublished data	Cautery pneumectomy and x-ray	A. K. No. 957 Died in 60+ days	? (no necropsy)

attempt at resection provided the extension is located peripherally. A biopsy if indicated should always be taken from the infiltrated tissue toward the hilus as suggested by Sauerbruch.

Exploratory Thoracotomy and Insertion of Radium.—If the exploratory operation shows that the tumor is not amenable to radical surgery, then direct implantation of radium or radon may be considered. Tudor Edwards has reported favorable results with this method. Wangensteen saw no benefit from radon in one case in which he implanted radon seeds. Radium therapy alone was attempted in one patient of the series to which we refer. A small incision was made in the axilla and four radium needles (total dose 50 mg., 20 hours) were inserted into the lung without exploration of the pleural cavity. The same dose of radium was repeated in twelve days but the patient died three weeks later. (See also Cases 29 and 30, Table IV).

Extirpation or Destruction of the Tumor.—The radical operative removal of cancer of the lung has been attempted both in Europe and America. The results so far are far from satisfactory, yet they hold out some hope that early diagnosis and improved surgical technic may eventually be rewarded by some cures. (Table IV.)

Five patients (Sauerbruch 2, Edwards 2, Allen and Smith 1) are known to be alive more than one year after the operation. The most successful cases so far reported are those of Sauerbruch. His patients were reported to be alive after three and five years. It is to be hoped that future reports will give more detailed information regarding the technic of the operation employed, the gross and histologic characteristics of the tumor removed, the cause of death in unsuccessful cases, and a more complete follow-up record.

Cautery Pneumectomy.—Seven patients with carcinoma of the lung have been treated by cautery pneumectomy at Barnes Hospital by Dr. Evarts A. Graham (Fig. 7). The operative mortality was 14.3 per cent. The purpose of the operation was to destroy the local growth and to provide drainage for areas of pulmonary suppuration. It was apparent from

the very beginning in some cases that the entire tumor could not be destroyed. Five of these patients lived 24, 25, 37, 90 days and 11 months, respectively, after the cauterization. Another patient, now dead, is known to have lived for at least 60 days. Three of the patients subjected to

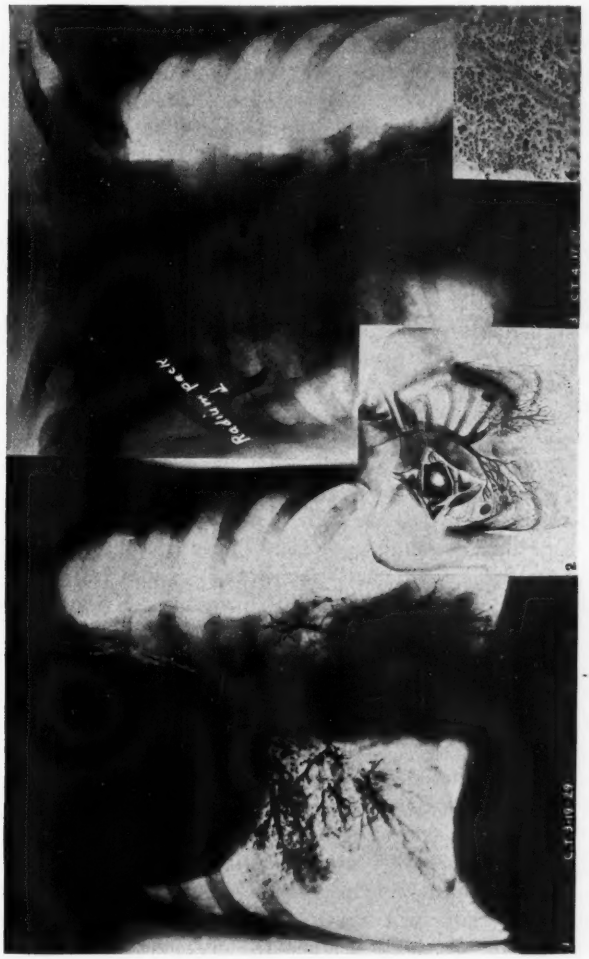

Fig. 7.—Primary carcinoma of the lung. The original film in this case suggested a mediastinal tumor. Note the displacement of the trachea and the fact that lipiodol failed to enter the right upper lobe. In Figure 2 one observes the evidence of increased venous pressure as manifested by collateral circulation, particularly in the right arm. This patient was subjected to a cautery pneumectomy when a radium pack was implanted (3). This patient eventually died from his disease. Necropsy in this case revealed a primary round cell carcinoma of the lung.

cautery pneumectomy are included in our necropsy series and the cause of death in each of the three was carcinoma of the lung with distant metastases.

Of the patients having cautery pneumectomy two had deep roentgen therapy also and two others had both radium and roentgen therapy but

these additional forms of treatment apparently did not influence the end result. The longest survival (11 months) occurred in a patient having deep roentgen therapy in addition to cautery pneumectomy. It is doubtful whether the slightly better result in this case can be attributed to the irradiation therapy.

Other Operative Procedures.—Radical operations for the removal of metastatic lung tumors have been carried out by Divis, Gerulanos and by Edwards. Lobectomy for sarcoma or lymphosarcoma has been reported by Helferich, Archibald and Eloesser. Operations for carcinoma of the lung of a less radical nature such as drainage of cavities or curetting out of tumor tissue have been performed by Macewen, Küttner, Körte, Eloesser, Lenhartz and others but are not included in the table because they do not constitute attempts at removal of the diseased area. Palliative decompression operations consisting of removal of ribs or a segment of the sternum are included in the reports of Sauerbruch and of Eloesser. We subjected one patient to an injection of his phrenic nerve and intercostal nerves for pain with slight temporary relief. This procedure is certainly deserving of further trial.

SUMMARY

Our observations do not permit us to draw any sweeping conclusions. They indicate rather where further attention may best be directed. The clinical history was found to be suggestive of carcinoma of the lung only when complaints referred to made their appearance in an individual of the cancer age. Pain was found to be more continuous than that due to any other intrathoracic condition. Dyspnea was due to a variety of factors, including increased intramediastinal pressure. Evidence which shows that the disease may run a relatively slow course in some instances, has nevertheless presented itself. The fact that 3 of the 26 patients who came to necropsy failed to show any regionary metastases is worthy of some consideration.

The physical findings called attention to the presence of a bronchostenosis, pulmonary consolidation or pleural effusions.

It is apparently possible for a bronchostenosis and its consequent atelectasis to occur without involvement of the regionary glands by tumor. This, however, constitutes an exception rather than the rule.

Fluoroscopy was found to be helpful in the diagnosis chiefly because it permitted one to determine whether the shadow present could be dissociated from the hilus and also because it enabled one to determine the condition of the corresponding diaphragm. The ordinary roentgen ray picture was on many occasions more than suggestive, and the lipiodol injection picture could seldom be called diagnostic per se.

Bronchoscopic examinations when employed were extremely helpful. The same could be said of biopsy. The examination of pleural fluid by the Mandelbaum method is recommended.

Three patients had no metastases at necropsy but in one of them a metastatic axillary node had been removed during life. In a second patient lobectomy would have been indicated but the diagnosis of carcinoma although suggested was not confirmed by an exploratory thoracotomy. The third patient died of other causes, the carcinoma being merely an accidental necropsy finding.

An attempt has been made to correlate the clinical findings of metastases with those observed at necropsy. The two findings often failed to coincide. The frequency of metastases to the kidney has been emphasized; also the importance of a complete examination of the genitourinary tract before undertaking any radical procedure.

The suggestion that an exploratory thoracotomy is indicated in the absence of clinical evidence of either regionary or generalized metastases seems well taken. This is occasionally the only means of establishing a diagnosis early. With regard to treatment, the results obtained by irradiation therapy have so far been extremely disappointing. Up to the present time the literature contains reference to only six malignant tumors of the bronchus which have been treated bronchoscopically. The results have been tabulated. The treatment of carcinoma of the lung by such operative procedures as lobectomy or cautery pneumectomy has so far been disappointing. Reference could only be found to 35 occasions on which such treatment was undertaken. The results have been tabulated. Cautery pneumectomy has the advantage of providing drainage in the presence of pulmonary suppuration and of having a relatively low operative mortality.

The implantation of radon seeds through the bronchoscope have produced fairly satisfactory results. The use of radon through an exploratory thoracotomy has received little attention. The combined form of treatment, however, appears to be deserving of further trial.

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We have included only some of the more important references which concern the surgical treatment of carcinoma of the lung. Other references have been intentionally omitted.

EXPERIMENTAL STUDIES ON THE EFFECT OF PARALYSIS OF THE DIAPHRAGM AND OF ITS REMOVAL*

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SINCE the recommendation of Stuertz[1] in 1911 the division of one or both phrenic nerves has become a widely accepted procedure in man for various pathologic conditions. The results have been satisfactory and without untoward effect. Harrington[2] has reported the use of phrenic evulsion as a palliative measure in cases of diaphragmatic hernia as well as an aid to the radical operative closure of such hernias. Portions of the paralyzed diaphragm have been used in plastic measures following removal of thoracic growths.

From the experimental viewpoint it is important to note that in 1890 Hare and Martin[3] cut one and both phrenic nerves in rabbits without causing death. Schlaepfer's[4] work (1923) seemed to settle the controversy that the phrenic nerve was the only motor nerve to the diaphragm in dogs.

Heuer and Holman[5] in 1923, following intrathoracic division of the phrenic nerve in dogs noted that passive motion of the paralyzed hemidiaphragm was present when the fluoroscopic examination was made in the erect position and also that the elevation of the paralyzed muscle was only slight. Examination in the recumbent position showed that the paralyzed diaphragm occupied a relatively high position and that it was practically immobile.

Lemon[6] in 1927 found that unilateral phrenicotomy did not change in any demonstrable way the movements or shape of the wall of the chest in dogs. The thoracic cavity was lessened in its long diameter and the pulmonary volume decreased by lessening one dimension only. Whether one or both phrenic nerves were divided, the dogs were competent to carry on the usual activities without embarrassment or dyspnea. The paralyzed hemidiaphragm remained stationary or had a slight movement or more frequently paradoxical movements.

Although circulation is entirely dependent upon muscular action, respiration is only partially dependent upon the diaphragm and is continued in a fairly satisfactory manner without its action.

In discussing the function of the diaphragm Lemon[7] stated: "the diaphragm therefore can be considered as having two functions: a primary and a secondary, the latter respiratory because by rhythmic contraction the thorax is enlarged in volume, and air is brought into the lungs, the

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former under more direct voluntary control producing pressure so that when opposed by the abdominal musculature the sphincters can be overpowered and the products of the intestinal, the renal, and the genital systems can be extruded.''

''Although the abdominal increase in pressure during the descent of the diaphragm tends to distend the abdomen, yet because there is opposition, the central tendon becomes a fixed point and the circumferential muscles in contraction actually elevate the lower ribs and expand the lower part of the thoracic cavity.'' He offered evidence in corroboration of that of others ''that the descent of the diaphragm actually increases the subcostal angle by increasing the intraabdominal pressure. This pressure exerted on the abdominal contents, which are comparable with a fluid, becomes hydrostatic in character and is transmitted equally in all directions, forcing outward not only the abdominal wall but the margins of the ribs.''

Hoover[8] in his experimental work believed that the contraction of the diaphragm narrowed the subcostal angle and tended to force the margins of the ribs toward the median line because when the diaphragm was paralyzed the subcostal angle was decreased.

Lemon[6] found at autopsy that in unilateral phrenicotomy the paralysis and uniform atrophy was a constant finding with the line of demarcation between the paralyzed and normal muscle in the midline and sharply drawn. The response to stimulation was lost throughout the whole of the affected hemidiaphragm. When light was transmitted through the whole diaphragm, on the paralyzed side the atrophy permitted the passage of light producing a translucency in contrast to the opaqueness of the normal side with its red muscular color. Histologically the muscle cells were reduced to a quarter of their normal size and fatty degeneration was observed in those of five months' duration.

A few months after the appearance of Lemon's article experiments were started to determine the late changes of unilateral phrenicotomy and especially to note whether hemiparalysis would produce any effect upon the growing puppy. Attempts were also made to produce diaphragmatic hernia and subsequently the effect of total paralysis of the diaphragm and the subtotal and total removal of the muscle was studied. The investigation of the paralysis and the excision of the diaphragm were undertaken to determine feasible procedures that might be applicable to man when pathologic conditions or growths required removal of a large portion of this muscle.

EFFECT OF UNILATERAL PHRENICOTOMY IN GROWING PUPS AND IN AN HYPOPHYSECTOMIZED PUP

Two litters of puppies, five and six weeks old, and one litter four months old were used. In each litter one animal was sacrificed at the time of phrenicotomy and the thoracic cage with attached diaphragm saved. The other members of each litter had an unilateral phrenicotomy. Either the

nerve was evulsed through an incision in the neck under ether anesthesia or a portion of the nerve as it coursed over the pericardium was resected by thoracotomy. One of the puppies was phrenicotomized and a week later when six weeks old the hypophysis was removed. Growth is inhibited following hypophysectomy so that the shape of the thoracic cage and the posi-

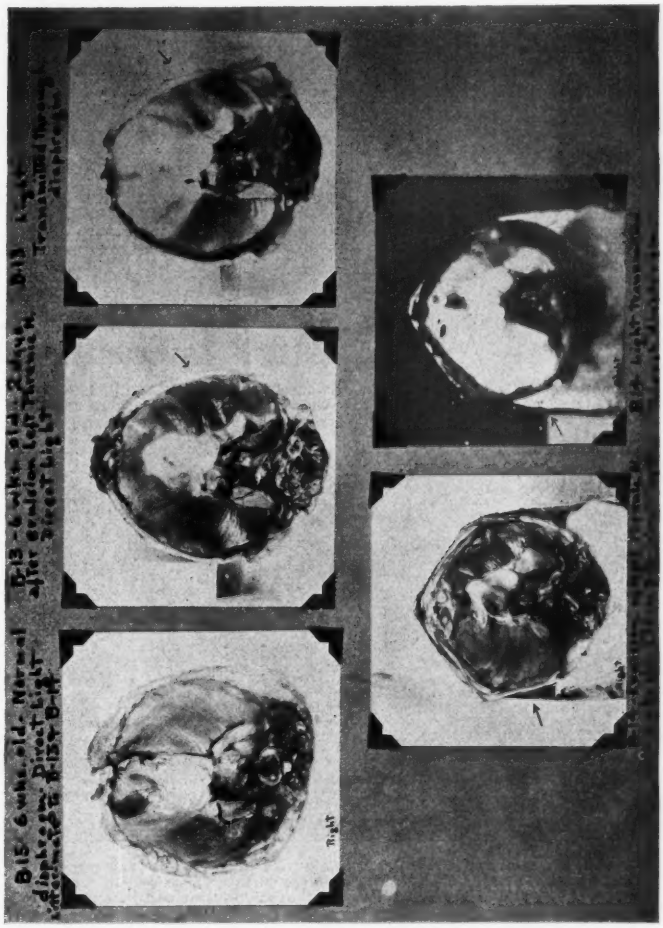

FIG. 1.—B_{13}, B_{14} and B_{15} were six weeks' old littermate puppies. The early changes in appearance of the paralyzed diaphragm noted on direct inspection and by transillumination are compared with the normal muscle of the littermate control.

tion and atrophy of its diaphragm in this animal in which no development occurred served as a means of comparison with its litter-mate in which only unilateral phrenicotomy had been performed. These puppies, P_4 and P_5, lived to be two years, two and a half months old. The period of survival in the other two litters was shorter.

In the six weeks old litter, B_{13}, B_{14} and B_{15} the survival period was 2 days and 17 days. The photograph of the diaphragms of these puppies (Fig. 1) indicated no alteration in the shape of the lower opening of the rib cage. The atrophy of the hemidiaphragm was evident on the second

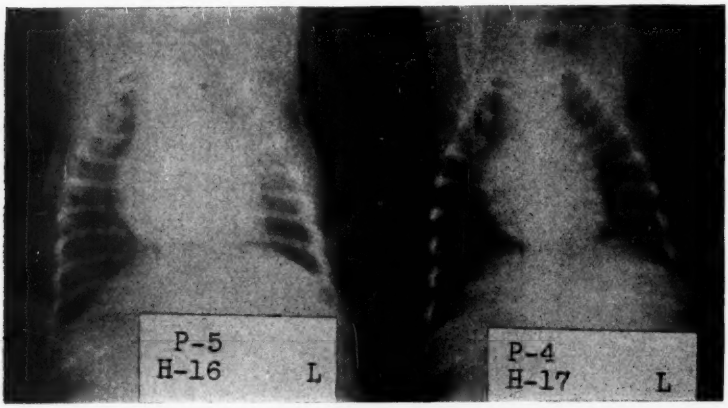

Fig. 2.—Roentgenograms of chests taken in recumbent position of P_4 and P_5 immediately after unilateral phrenicotomy when five weeks old showing the high position of the left diaphragm on inspiration.

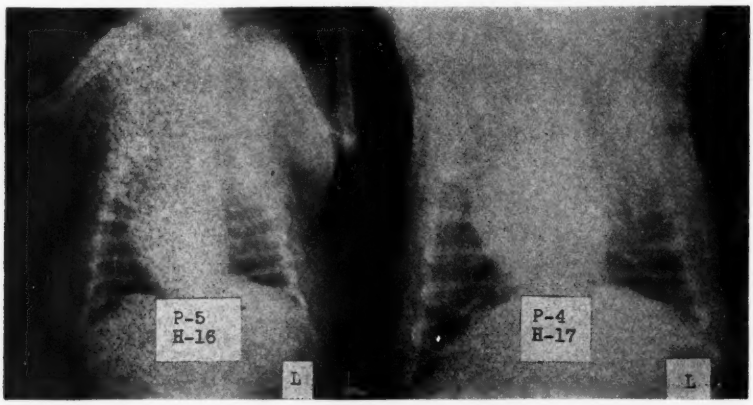

Fig. 3.—Roentgenograms of chests of P_4 and P_5 six months after left phrenicotomy when seven and one-half months old. P_5 was also hypophysectomized when six weeks old so that growth was inhibited. No asymmetry in thoracic outline can be seen. The left diaphragms remain elevated.

day and quite marked by the seventeenth day when on transillumination the paralyzed side was translucent.

A survival of 24 days, 48 days and 15 months after unilateral phrenicotomy was secured in the four months old litter. During the period of observation no change was appreciable in the growth and development of the thorax. The respiratory excursions seemed equal and when sacrificed the

hemiatrophy of the diaphragm was complete with a sharp line of demarcation from the normal muscle.

The most interesting experiment of this series was with the five weeks

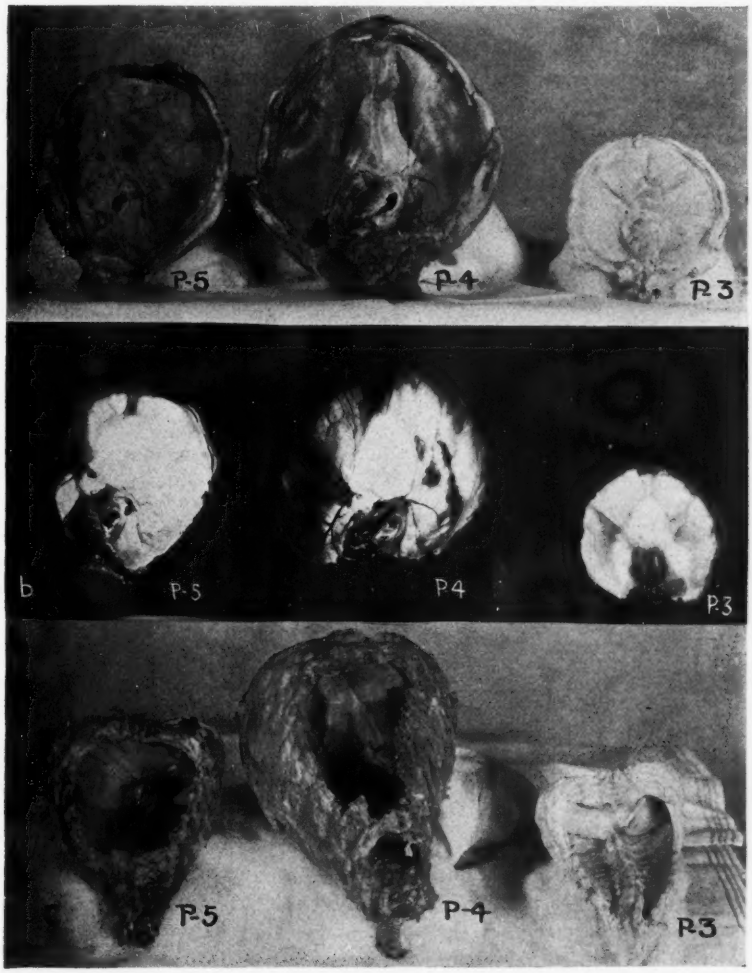

Fig. 4.—*A.* photograph of lower thoracic outlet of P_3, sacrificed when five weeks old to compare the size of the normal diaphragm with those of its littermates P_4 and P_5 which had a left hemiparalysis for ever two years. *B.* Diaphragms of P_3, P_4 and P_5 photographed by transmitted light. *C.* Photograph of thoracic cages of P_3, P_4 and P_5. Note the high portion of atrophied diaphragm in P_5, the hypophysectomized animal.

old litter (P_3, P_4, P_5). One puppy, P_3, was sacrificed at this age and the thoracic cage with attached diaphragm preserved while P_4 and P_5 had their left phrenic nerve evulsed. One week later when six weeks old P_5 was

hypophysectomized which inhibited her growth. Roentgenograms (Fig. 2) taken in the recumbent position on inspiration with the tube at six feet distance showed the high position of the left diaphragm and outlined the thoracic cage. At this time P_5 was somewhat larger than her littermate. Six months later when she was seven and a half months old and when P_4 had attained adult life, as indicated by closure of the epiphyses of the long bones, the roentgenograms (Fig. 3) showed no asymmetry of the thoracic cage. The left diaphragms were high, with a higher dome in P_5. The position of the heart in both animals seemed shifted slightly to the right compared with the earlier roentgenograms. Respiratory excursions were equal

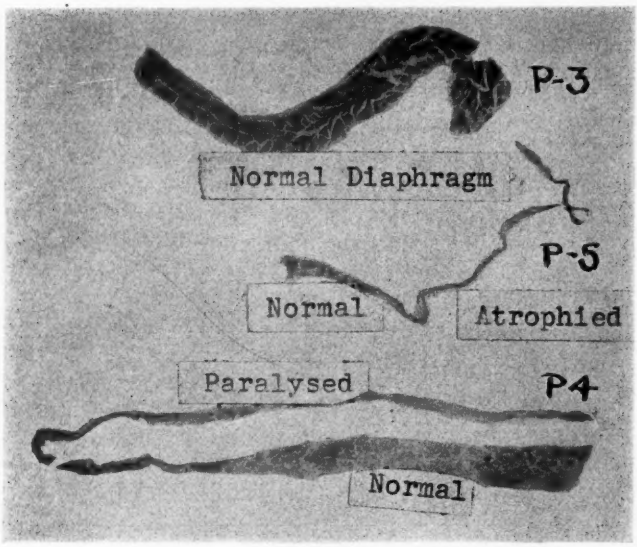

Fig. 5.—Sections of diaphragms of P_3, P_4 and P_5 magnified 4×. Sections of P_4 and P_5 include the normal and atrophied areas.

throughout the period of observation and one was unable to ascertain on inspection whether or not phrenicotomy had been performed.

At autopsy, over two years after unilateral phrenicotomy the hemiatrophy of the diaphragm in the normally developed puppy P_4 extended to the midline, but in the hypophysectomized animal P_5 it seemed to extend beyond the midline especially in the region of the dome (Fig. 4, *A*), which caused the atrophic portion to extend high into the thoracic cage (Fig. 4, *C*). This was quite evident on transillumination (Fig. 4, *B*). The outline of the thoracic cage seen from below in P_4 and P_5 was as symmetrical as in P_3 sacrificed when five weeks old.

Histologically, there were occasional areas with faint outlines of muscle cells with fatty degeneration but most of the atrophied portion was made

up of fibrous tissue and much thinner than the muscular normal side (Fig. 5).

Summary.—Unilateral phrenicotomy performed in puppies five and six weeks old as well as when half grown and observed as long as two years after operation, caused no change in any demonstrable way in the movement or shape of the thorax. Development of the thoracic cage was normal compared with that of a littermate sacrificed at the time of operation and compared with that of a littermate hypophysectomized puppy (growth thereby inhibited) which also had a unilateral phrenicotomy.

ATTEMPTS AT THE PRODUCTION OF DIAPHRAGMATIC HERNIA WITH AND WITHOUT PREVIOUS HEMIPARALYSIS OF THE MUSCLE

Traumatic diaphragmatic hernia in man may be produced by sudden pressure or force upon the abdominal contents thereby causing a tear in the normal muscle or in a congenitally weak portion of the muscle. In order to effect its production in the dog without causing suffering, the tear in the paralyzed or normal left hemidiaphragm was made under ether anesthesia. At operation through the opening in the upper abdomen a stout linen thread was passed through the dome of the left diaphragm in such a manner that it would cut a crescent or nearly circular opening. The ends of the thread were brought out through the incision which was then tightly closed in layers. By a sawing motion on the protruding ends of the thread it cut through the diaphragm and then was pulled out of the abdomen. With the animal still under ether anesthesia sudden pressure was made upon the abdomen. In some instances the abdomen was distended by the intraperitoneal injection of air before the application of force. Fluoroscopic and roentgenographic examinations were made to determine the extent of the hernia.

In a four months' old puppy with a left diaphragm paralyzed for a month, a nearly circular tear over one centimeter in diameter was made with the linen thread. Following closure of the wound sudden pressure on the abdomen was made. Roentgenograms showed a peaked left diaphragm with the heart pushed over to the right. Two weeks later a second laparotomy was done. A portion of the liver had sealed the diaphragmatic tear. A linen thread was passed through the left lateral portion of the paralyzed muscle well away from the liver. Sudden pressure was again made on the abdomen after closure of the wound. Roentgenograms revealed no change from that noted previously. A third exploration was performed two weeks later. The stomach and liver were found adherent to the rent in the diaphragm. Again the thread was sutured into the muscle so that a semicircular tear three or more centimeters in diameter would result. After abdominal closure 40 c.c. of air was injected intraperitoneally and sudden pressure was applied. This time on roentgenographic examination a herniation about two by three centimeters was seen. Five months later a litter of seven puppies was born. At autopsy six months

later a small nubbin of omentum, two or three centimeters in diameter and protruding into the thoracic cavity three centimeters, was found in the rent of the atrophied hemidiaphragm. A thin, transparent sheet of tissue, probably peritoneum, separated the contents of the hernia from the lung which was not adherent.

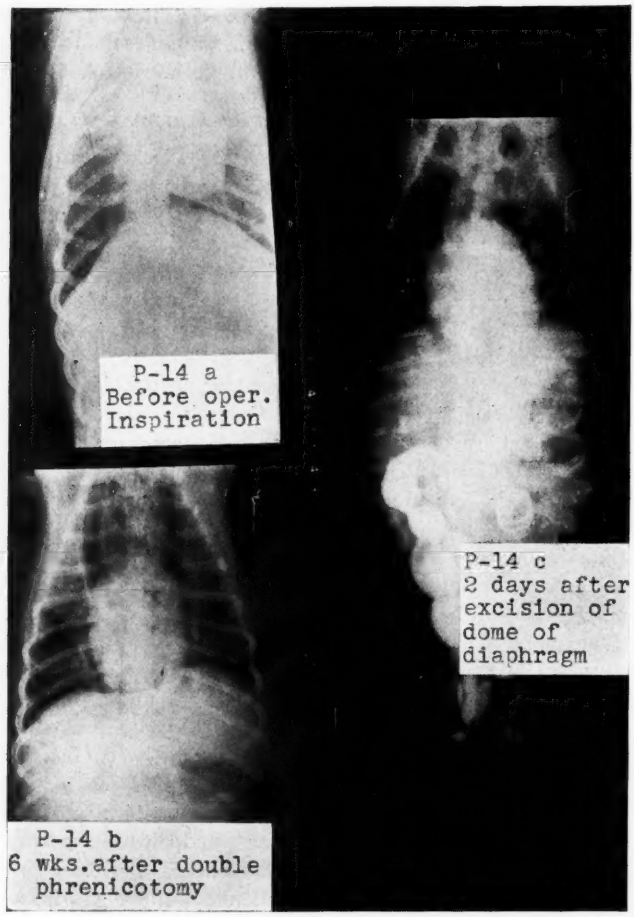

Fig. 6.—a. Roentgenograms of P_{14} on inspiration before any operative procedure. b. Six weeks after bilateral phrenicotomy showing elevated, paralysed diaphragm and the midthoracic enlargement from widened and elevated lower ribs and from the ascent of abdominal contents. c. Barium meal given two days after excision of both domes of the diaphragm indicating further ascent of abdominal viscera.

In an adult dog with a normal diaphragm two tears were made on the left side near the costal margin. Roentgenograms after sudden abdominal pressure showed no change. Two weeks later on exploration the tears were found sealed by liver and adherent lung. The thread was sutured along

the costal margin for a distance of four to five centimeters. After closure the abdomen was distended firmly with air and sudden pressure was alternately applied to the chest and to the abdomen. No herniation occurred and nine months later at autopsy the stomach was adherent to the rent sealing it off.

Since herniation could not consistently be produced in this manner, deliberate excision of both domes of a paralyzed diaphragm was performed on a dog, P_{14}. The crura as well as the esophageal hiatus and the vena cava opening were not disturbed. The bilateral phrenicotomy six weeks previously had lead to a sunken abdominal wall with paradoxical respiration. Following excision of the domes the abdomen became more concave. Roentgenograms (Fig. 6, *A, B, C*) revealed the domes at a level one to two ribs higher. A barium meal showed the left dome to be formed by stomach. At laparotomy three months later the right dome was identified as liver and the left as stomach. No hernial sac had formed and the few fine adhesions when divided freed the liver and stomach leaving the thoracic cavity widely open. Following the bilateral phrenicotomy and further exaggerated by the herniations, the lower eight ribs were elevated causing a widening of the interspaces and an enlarging of the midportion of the thoracic cage as well as increasing slightly the subcostal angle. With the diaphragm functionless a certain amount of antagonistic action was eliminated so that the abdominal contents moved higher into the thoracic cage. This shift of abdominal contents along with the action of the intercostal muscles elevated the ribs and thereby considerably increased the circumference of the midthorax. The increased volume in this midthoracic region compensated in a moderate degree for the decreased volume produced by the elevation of the paralyzed diaphragm as well as by the hernial contents.

Three months after excision of the domes the roentgenographic picture had changed in that the thorax had become narrower with lessened subcostal angle, absent midthoracic enlargement and more angulated ribs. The pulmonary area was slightly decreased by this alteration in outline of the thoracic cage, although there was no further ascent of abdominal contents.

Summary.—It was exceedingly difficult to produce diaphragmatic herniation in dogs by tears in the muscle and sudden abdominal pressure. Even the large herniation following excision of the domes of the paralyzed diaphragm encroached very little upon the thoracic volume.

EFFECT OF DOUBLE PHRENICOTOMY WITH TOTAL PARALYSIS OF THE DIAPHRAGM

Five animals were observed over a period of two to eleven weeks after bilateral phrenicotomy. They were subsequently used for studies of subtotal or total diaphragmectomy. All except one (P_{18}) at once developed and maintained paradoxical respiration following intrathoracic resection of the phrenic nerves. The exception developed postoperative distemper pneumonia with multiple infarcts. The rapid respirations, the pleural

exudate and the consolidated lower lobes made it difficult to determine the type of respiration. The animal was sacrificed two weeks after operation.

The remaining four animals had a paralyzed diaphragm from four to eleven weeks. They quickly became accustomed to the paradoxical respiration and were as active as normal dogs. The abdomen rapidly became scaphoid. Within a few weeks the anterior abdominal wall in the region of and below the umbilicus appeared to lie against the vertebral column. The abdominal contents had ascended and only an occasional loop of small bowel and the descending colon could be palpated in the lower abdomen. Coincident with the sinking of the abdominal wall the circumference of the lower end of the thoracic cage increased slightly, widening the subcostal angle, while in the midthoracic region the bulging of the cage was quite noticeable. This midthoracic increase in circumference was explained by the ascent of the abdominal contents into this region with the elevation of the paralyzed, passive and functionless diaphragm and by the unantagonized, unopposed action of the intercostal muscles elevating and separating the lower ribs. This enlargement of the thoracic cage at about the level of the dome of the diaphragm could be determined with a certain degree of accuracy by measurements of the pulmonary area in the roentgenograms of the chest taken at a standard distance of six feet at the height of inspiration (Figs. 7 *A* and *B*). This midthoracic enlargement roughly compensated by half the decrease in pulmonary area due to the ascent of the paralyzed diaphragm. The slight widening of the lowermost portion of the thoracic cage with the increased subcostal angle apparently had no effect on the size of the pulmonary area.

Roentgenograms taken at the height of expiration (Figs. 7 *A* and *B*) showed the thoracic cage to be the same size before and after double phrenicotomy, but the pulmonary area was slightly decreased since the paralyzed diaphragm remained elevated. In other words the maximum shrinkage of the pulmonary area on inspiration after diaphragmatic paralysis was roughly decreased by a half due to the midthoracic enlargement.

The cardiac shadow (Fig. 7, *A* and *B*) in the roentgenograms taken at the height of inspiration was slightly enlarged and shifted to the right after diaphragmatic paralysis. In the normal dog the cardiac area was found to be slightly smaller at the height of inspiration than at the height of expiration. The slight decrease in size may be explained by the fact, as Lemon[7] stated, that with the contraction of the diaphragm intraabdominal pressure was increased which exerted pressure on the vena cava forcing blood into the thorax and heart, but before inspiration was completed the venous flow was lessened and the blood pressure lowered resulting in a smaller cardiac shadow. With diaphragmatic contraction eliminated by double phrenicotomy intraabdominal pressure on the vena cava is continued throughout inspiration resulting in a slightly larger cardiac shadow compared with expiration. The cardiac area on expiration remained the same whether the diaphragm was paralyzed or not.

Summary.—In the normal dog roentgenographic evidence indicated that on inspiration the outlet of the thorax and the subcostal angle were increased compared with expiration. The cardiac shadow at the height of inspiration was slightly smaller than at expiration. Following double phrenicotomy with diaphragmatic paralysis paradoxical respiration at once developed. With the sinking in of the abdominal wall the lower thorax and especially the midthoracic region enlarged on inspiration which compensated roughly by half the decrease in pulmonary area caused by the

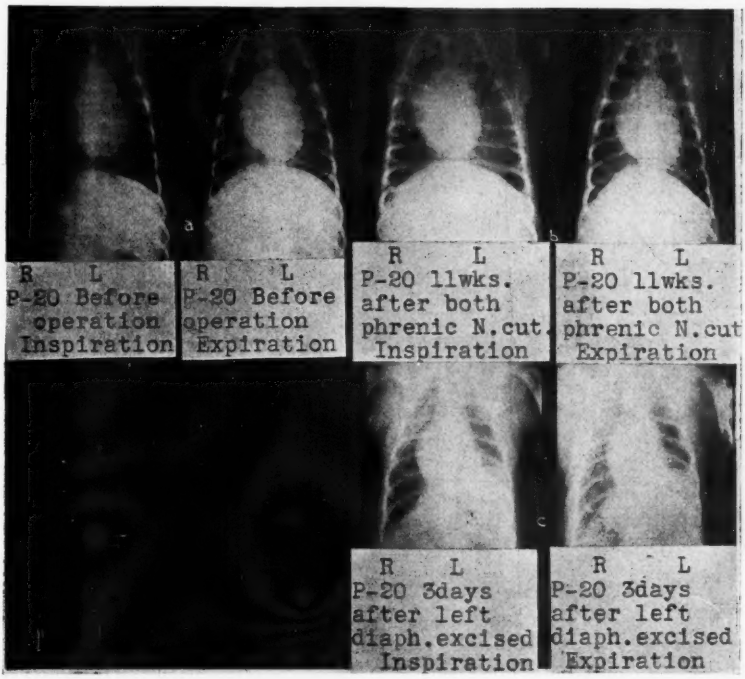

Fig. 7.—Three sets of roentgenograms of P_{20} taken at height of inspiration and of expiration. *A.* Before any operative procedure. *B.* Eleven weeks after bilateral diaphragmatic paralysis. *C.* Three days after subtotal diaphragmectomy. Note the midthoracic enlargement, the elevated diaphragm, widened and elevated lower ribs and slightly enlarged cardiac shadow on inspiration after subtotal diaphragmectomy.

elevated diaphragm. The pulmonary area showed an absolute decrease on inspiration after paralysis but the maximum effect was offset by the midthoracic enlargement.

On expiration the outline of the thoracic cage was the same as before diaphragmatic paralysis but the pulmonary area was decreased only by the elevation of the diaphragm.

The cardiac shadow after diaphragmatic paralysis was slightly larger and shifted to the right on inspiration compared with expiration.

EFFECT OF TOTAL AND SUBTOTAL DIAPHRAGMECTOMY

The purpose of these experiments on seven animals was to determine the amount of diaphragm that could be removed successfully; whether previous paralysis of the muscle facilitated its removal; and what factors jeopardized a successful operative procedure. It was hoped that information might be obtained by these experiments as to the feasibility of excision of large portions of the human diaphragm for malignant growths.

The operative approach for diaphragmectomy in the dog was by a curved incision through all tissues from one anterior axillary line to the other, parallel and about two centimeters below the costal margin. Division of the abdominal muscles through this incision did not interfere with

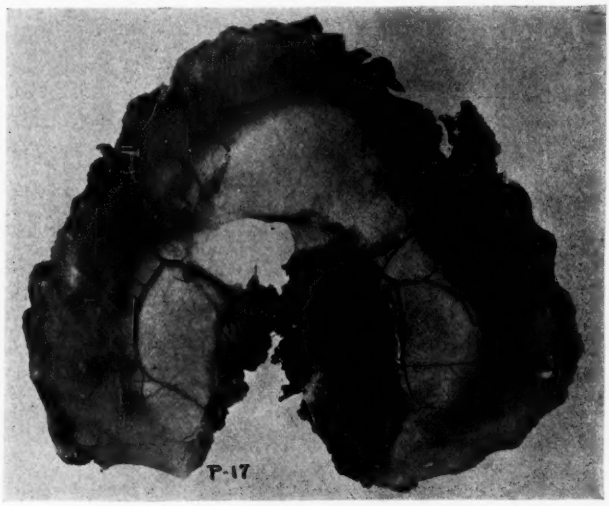

Fig. 8.—Photograph of excised diaphragm of P_{17}.

their nerve supply. Before the diaphragm was cut artificial respiration was started through the tracheal tube. All bleeding points were tied or transfixed with silk and the abdominal closure in layers was carefully made with double medium continuous silk to the peritoneum and posterior sheath, double medium figure-of-eight stitches to the fascia of the muscles, and interrupted fine silk stitches to the subcutaneous layer and skin.

Diaphragmatic paralysis preceding total removal was applied in animals P_{14}, P_{17} and P_{19}. In P_{14} after paralysis herniation was produced by excision of both domes of the muscle. Two months later the entire diaphragm was excised by division of the circular attachment to the thoracic cage, section of the crura, freeing of the esophagus and vena cava and mobilization of the mediastinum at its attachment to the diaphragm. While handling the stomach, food was regurgitated and some of it aspi-

rated. Following operation the paradoxical respiration became more marked and the abdominal wall more sunken. The next day pneumonia, apparently from aspiration, was noted and on the second postoperative day death occurred. Autopsy showed considerable hemorrhage in the thoracic cavity and extensive pneumonia.

Roentgenograms taken just before and shortly after diaphragmectomy revealed no further elevation of the abdominal contents on inspiration compared with the preoperative hernial level, but on expiration the level on the left was over a rib higher, with the level on the right remaining the same. The midthoracic diameters remained the same with a greater circumference on expiration compared with inspiration.

Six weeks after double phrenicotomy in an adult fox terrier, P_{17}, total excision of the diaphragm was performed as in P_{14} (Fig. 8). The lower

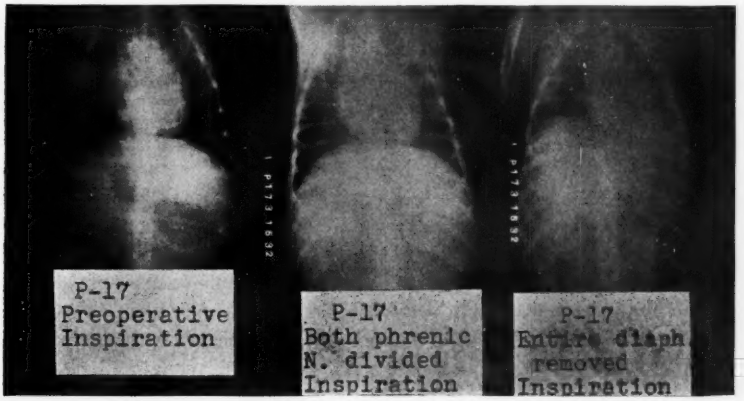

Fig. 9.—Roentgenograms of P_{17} taken on inspiration just before and six-weeks after bilateral phrenicotomy, and immediately after total diaphragmectomy. Note the narrowed sunken abdomen after diaphragmatic paralysis.

end of the mediastinum, which was freed from its attachment to the diaphragm, was sutured to the anterior chest wall in the zyphoid region. This immobilization of the mediastinum stabilized the heart and changed a slow irregular action to strong regular contractions. Care was taken as the abdominal wound was closed not to overinflate the lungs. The dog died during the night and at autopsy the next day death was found to have been caused by venous hemorrhage from unsecured diaphragmatic branches of the vena cava. Roentgenograms (Fig. 9) made just before and after total diaphragmectomy showed on inspiration that the level of the abdominal contents had risen two to three ribs higher. The ascent of the liver displaced the heart to the left. The pulmonary area was considerably decreased. The outline of the thoracic cage remained the same as after paralysis of the diaphragm.

Another adult fox terrier, P_{19}, had a total diaphragmectomy seven

weeks after bilateral phrenicotomy. A tape occluded the cardiac end of the esophagus to prevent regurgitation during the removal of the muscle. When the mediastinum was freed from the diaphragm the heart rate slowed to below forty per minute but improved when the mediastinum was sutured to the anterior thoracic cage. The animal died 22 hours after operation. Large amounts of dark blood were passed by stool before death. At autopsy a small amount of blood was found free in the abdomen. The liver was deep red, firm and markedly congested on section. A few infarcts were seen in the spleen which was deep red. The kidneys showed marked venous changes and in the large bowel the mucosa was hemorrhagic and gross blood was found in the colonic contents. The lower pulmonary lobes were collapsed. The venous congestion was due to rotation of the mobilized liver and kinking of the vena cava, though at autopsy the latter was found patent.

Total diaphragmectomy was performed on two dogs without previous paralysis of the muscle. One animal, P_{16}, died during abdominal closure when the breathing became shallow and the pulse markedly slowed. Artificial respiration was of no avail. In the other dog, P_{15}, the mobilized mediastinum was not anchored so that the heart rate remained slowed. Artificial respiration was maintained at intervals for three hours. It would return the normal color to the mucous membranes, the corneal and other reflexes appeared, spontaneous limb movements were noted, but when it was stopped spontaneous breathing continued five or six times then became labored and ceased. The animal became comatose, pale and reflexes were lost. The heart beats were feeble and infrequent. Artificial respiration soon returned the animal to the semiconscious state again. When sacrificed in three hours the liver was greatly congested, the vena cava kinked because of the shift in the position of the liver and the heart quite moveable.

These experiments of total diaphragmectomy clearly indicated that it was not the removal of the entire muscle that caused death but disturbance of the position of the heart when the mediastinum was separated from the diaphragm, and of the vena cava when it was freed from its diaphragmatic hiatus that markedly impaired the circulation and resulted fatally. The heart beat became slow and irregular with the mobilization of the mediastinum but returned to a fairly normal state when the mediastinum was fixed and sutured. The mobilized vena cava and liver led to kinking of this great vessel. The portal vein was likewise affected so that fatal passive congestion occurred in the vital abdominal organs.

It was thought that if the cardiac position could be fixed by suturing the mediastinum to the chest wall and angulation of the thoracic vena cava prevented by preserving its diaphragmatic hiatus, then excision of the rest of the diaphragm, subtotal diaphragmectomy, might be successful.

This subtotal diaphragmectomy was attempted on two dogs. One ani-

mal, P_{20}, had the muscle paralyzed eleven weeks previously. In the other dog, P_{21}, the operation was performed on the normal diaphragm.

Over two-thirds of the paralyzed diaphragm was removed in P_{20}. Both crura were excised, the esophagus and stomach freed, and the mediastinum sutured to the anterior thoracic wall. The hiatus of the vena cava in the diaphragm was not disturbed nor was the diaphragmatic attachment along the right costal margin. The animal recovered from the anesthetic without any difficulty. The paradoxical respirations continued and seemed more pronounced. Roentgenograms (Fig. 7, *C*) taken three days after operation revealed the small portion of remaining diaphragm to be at about the same level as after paralysis of the muscle. On the left side the abdominal contents had ascended three ribs higher. The midthoracic enlargement noted on inspiration after double phrenicotomy was not apparent after subtotal diaphragmectomy. On expiration the thoracic outline remained the same as before and after double phrenicotomy. In contrast to the midthoracic enlargement on inspiration compared with expiration noted after diaphragmatic paralysis, following subtotal diaphragmectomy, the thoracic and pulmonary areas remained about the same, yet respiration did not seem embarrassed.

The abdominal wound became infected and on the seventh postoperative day broke down causing pneumoperitoneum and fatal pneumothorax. The lungs, vena cava, liver, spleen, kidneys and intestines appeared normal. The left two-thirds of the liver and the stomach were in the thoracic cavity. From observations of the animal one felt that survival would have continued if the wound had not suppurated causing pneumothorax.

In a young adult mongrel, P_{21}, all of the diaphragm except a narrow strip along the right costal margin, which extended to and included the hiatus of the vena cava, was excised. The severed end of the mediastinum was attached to the sternum. Paradoxical respirations developed immediately after operation. Gradually the abdominal wall became more and more sunken. The subcostal angle by roentgenographic measurements (Fig. 10) was gradually decreased, the lowest ribs seemed drawn to the median line and the midthoracic region was less in circumference than normally, both on inspiration and expiration. The thoracic cage became shorter and narrower since the ribs did not become elevated and separated but lay closer together and were more angulated in both phases of respiration. Curiously, immediately after operation the contraction of the strip of normal diaphragm along the right costal margin pulled downward the right border of the liver, kept the vena cava stretched, and increased the right pulmonary area by a distance of two to three ribs. The left pulmonary area was decreased over a third by the ascent of the abdominal contents which also displaced the heart to the right.

Two and four weeks after operation (Fig. 11) the roentgenograms showed continued ascent of abdominal contents, mainly portions of the in-

testinal tract, on both sides. After four weeks on the right side the abdominal contents had again reached the preoperative level. On the left side the contents were over five ribs higher, so that on inspiration the pulmonary area on the right was roughly decreased one-half and on the left

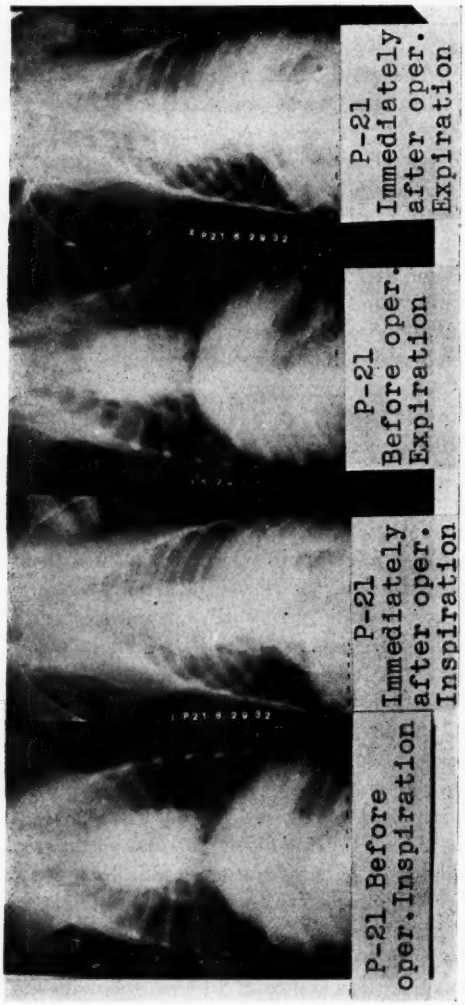
P-21 Before oper. Inspiration
P-21 Immediately after oper. Inspiration
P-21 Before oper. Expiration
P-21 Immediately after oper. Expiration

Fig. 10.—Roentgenograms of P_{21} taken at the height of inspiration and expiration before and immediately after subtotal diaphragmectomy, showing narrow, shortened thoracic cage in contrast to the effect after diaphragmatic paralysis (Figs. 7, 9). The contracted strip of remaining diaphragm has enlarged the right pulmonary area.

three-fourths. On expiration the decrease on the right was about two-thirds and on the left over three-fourths.

The dog remained in good condition for a month and seemed to be a little more inconvenienced than after diaphragmatic paralysis. Distemper

then developed with anorexia and rapid respiration. When the distemper became severe the animal was sacrificed five weeks after operation.

When the thorax was opened and the contents undisturbed, viewed from the left side the collapsed pulmonary lobes permitted one to see that the small bowel occupied the anterior median portion of the left chest nearly

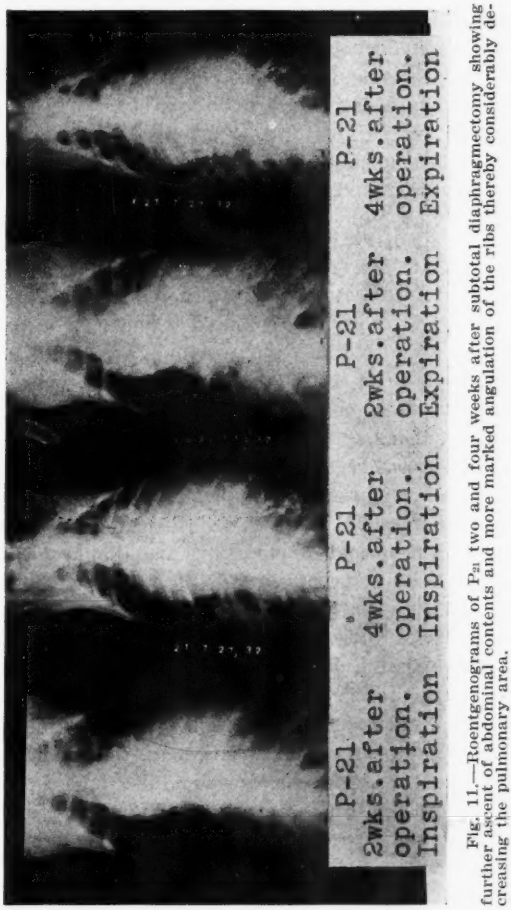

Fig. 11.—Roentgenograms of P_{21} two and four weeks after subtotal diaphragmectomy showing further ascent of abdominal contents and more marked angulation of the ribs thereby considerably decreasing the pulmonary area.

to the apex. On the right side above the small strip of diaphragm, was the stomach pushing up the heart. Anteriorly were loops of intestines with large bowel next to the stomach. Further exposure on this side showed the straight course of the thoracic vena cava, the strip of diaphragm, the collapsed right pulmonary lobes and the stomach pressing upward the heart. All surfaces were smooth and glistening. There was no evidence

of infection except the distemper bronchopneumonia in the right lower lobe.

Summary.—Removal of the entire diaphragm with or without previous paralysis was unsuccessful due to mobilization of the heart produced by separation of the mediastinum and diaphragm, and by kinking of the vena cava causing marked congestion of vital abdominal organs. When the heart was immobilized by anchoring the mediastinum to the anterior chest wall and when the vena cava maintained its normal course by leaving its diaphragmatic hiatus undisturbed, subtotal diaphragmectomy seemed to disturb the animals no more than after diaphragmatic paralysis or diaphragmatic herniation. Unfortunately wound infection and distemper interrupted possibly long survival periods although one animal remained in good condition until the fifth postoperative week when fatal distemper pneumonia developed.

CONCLUSIONS

1. Unilateral phrenicotomy, performed in puppies five and six weeks old as well as when half grown and observed as long as two years after operation, caused no changes in any demonstrable way in the movement or shape of the thorax. The development of the thoracic cage was symmetrical and in one litter containing a normally developing phrenicotomized pup and another pup phrenicotomized and hypophysectomized to inhibit growth, the thorax remained unchanged.

2. Paralysis was immediate and complete hemiatrophy evident within two weeks after unilateral phrenicotomy.

3. Tears made in a normal or paralyzed hemidiaphragm followed by sudden abdominal pressure generally failed to produce herniation. Even when the dome of the paralyzed diaphragm was excised the roentgenographic studies showed little early change except a higher level of abdominal contents compared with simple paralysis of the diaphragm, yet after three months the thorax had become narrow with angulated ribs, absence of midthoracic enlargement and slight decrease in pulmonary area.

4. Immediately following diaphragmatic paralysis by bilateral phrenicotomy, paradoxical respiration developed and the abdomen became markedly scaphoid, yet activity of the animals seemed unimpaired.

The lower thorax and especially the midthoracic region enlarged on inspiration which compensated roughly by half the decrease in the pulmonary area caused by the elevation of the paralyzed diaphragm.

On expiration the outline of the thoracic cage remained the same as before paralysis, but the pulmonary area was decreased only by the elevated diaphragm.

After paralysis the cardiac shadow on inspiration was slightly larger and shifted to the right.

5. Total removal of the diaphragm, whether previously paralyzed or not, was uniformly fatal.

It was not the removal of the septum between the thorax and the abdomen that was disastrous but the interference with circulation from the mobilized heart and thoracic vena cava.

Even when the heart was immobilized by suturing the divided mediastinum to the anterior thoracic cage, kinking of the vena cava caused the passive congestion of the vital abdominal organs.

6. Subtotal diaphragmectomy was successfully performed provided the mediastinum was sutured to stabilize the heart and provided the thoracic vena cava was left undisturbed by leaving a strip of diaphragm including the hiatus of this great vein attached to the thoracic cage.

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HERNIA OF THE LUNG*

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HERNIA of the lung is evidently extremely uncommon as one must presumably conclude from its infrequent mention in current surgical literature and its rare occurrence in the personal experience of even those especially interested in thoracic surgery. Yet it was most surprising to find from a careful study of all available literature that there really were as many cases as reported in this paper. This communication will not include mediastinal hernia, because it is felt that this type of hernia should be considered separately.

According to available literature, hernia of the lung was first reported by Rolland in 1499. Similar cases were described by Plater in 1461, by Boerhaave in 1709 and by Storch in 1750. According to Hertzberg (quoted from R. Wahl) the reports of Fabricius, Hildanus, Tulpins and Ruysch are even of older date. All these cases can hardly, however, be designated as true pulmonary hernias since they can all be traced to perforated wounds of the thorax following which a prolapse of the lung occurred. The first basic work on pulmonary hernias was undertaken by Morel-Lavallee. In 1849 he compiled a detailed study of cases, described the symptomatology and classified the various types of pulmonary hernias. It appears that from a study of the literature, cases reported as hernia of the lung were not all true hernia. Montgomery and Lutz were able to report 165 cases prior to 1925 (until 1800, 15 cases were reported, probably only 9 of these, they state, were true lung hernias). To date 171 cases have been found reported in the literature.

A true hernia of the lung is a protrusion of the lung beyond its normal boundaries through an abnormal opening in the thoracic enclosure. The sac usually consists of parietal pleura and is often firmly attached to the overlying skin.

The apparent rarity of this condition warrants the report of another case:

CASE No. 28034.—A rather emaciated seaman, forty-two years of age, entered the Broad Street Hospital on January 17, 1932, giving a family history of minor importance.

The patient's past history showed that when six years old, he was gored by a bull on the right side of the chest. The patient was unable to state whether at the time of the injury a fractured rib or a wound was present.

Shortly after the accident, a soft swelling appeared on the chest wall and this swelling persisted up to the time of admission to the hospital. The swelling had slowly but progressively enlarged without any associated pain or discomfort. For a considerable time he had been expectorating a large quantity of fetid sputum amounting to about

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10 ounces in 24 hours. The patient was not aware of any evening rise in temperature. He never had any hemoptysis or night sweats and had not lost weight. He did, however, become rather dyspneic on exertion.

Examination of the head, the oral cavity and sinuses did not disclose any pathologic findings.

The chest was emphysematous; supra and infraclavicular fossae were quite deep on each side. The costal angle was obtuse.

On the right side, below and lateral to the nipple in the fifth intercostal space, there was a retraction of the skin and underlying tissue during inspiration. This bulged out during expiration, reaching the size of an orange during forced expiration. That tissue mass seemed soft to palpation and manipulation; on auscultation it was crepitant. The remainder of the pulmonary area showed diffuse sibilant râles on both sides at the bases.

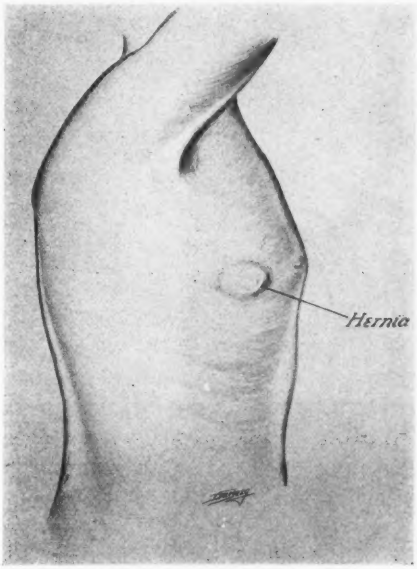

Fig. 1.

The heart was normal. Superficial and deep reflexes were present and normal.

Admission Diagnosis: An oval defect was noted between the fifth and sixth ribs in the anterior axillary line, measuring 3½ by 2½ inches, with adjacent lung and overlying tissue herniating through this bony defect.

Laboratory Findings.—Blood Count: Hemoglobin, 88 per cent; R. B. C., 4,750,000; W. B. C., 8,200; polys, 68 per cent.

Urinalysis: Moderate number of hyaline casts. Faint trace of albumin.

Wassermann: Negative.

Kahn Test: 2 plus. Sputum negative for T. B.

The patient was operated on by the writer on January 18 under local anesthesia. A curved incision was made from the midaxillary line extending forward along the lower border of the sixth rib and then curving upward to meet the lower border of the fifth rib. The intercostal space between the fifth and sixth ribs was found to be widened anterior to the anterior axillary line. The hernial sac was isolated and consisted of

pleura which was firmly adherent to the overlying skin. The hernial sac was oval and measured about 4 inches at its longest diameter and 3 inches across its widest portion. No adhesions were found in the sac. The lung was greyish and contained many emphysematous blebs. The redundant pleura was then resected and the pleural cavity closed with continuous suture. In exposing the fifth and sixth ribs some muscle tissue and fat were purposely left attached to the periosteum. The periosteum of the fifth rib was in-

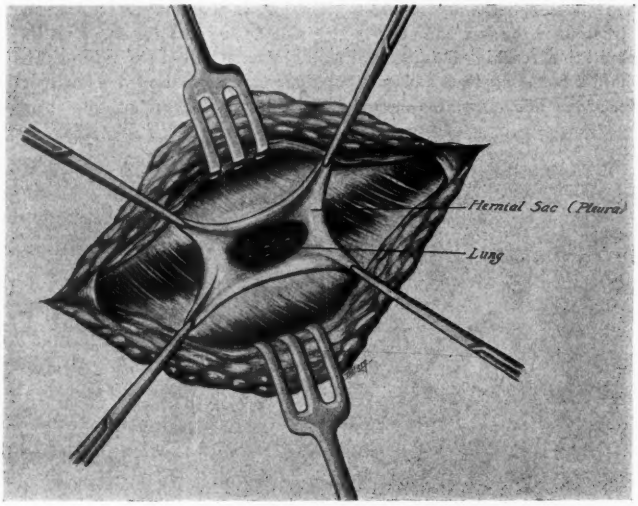

Fig. 2.

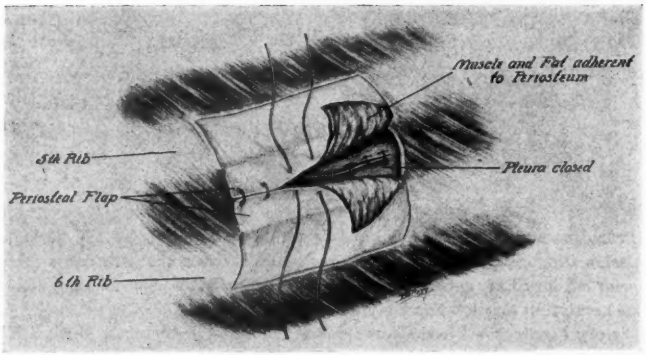

Fig. 3.

cised at the upper border of this rib and stripped down to the lower margin, and then the periosteum of the sixth rib was stripped up toward the fifth rib after incising it along its lower border. The two periosteal flaps were then turned in so that muscle and fat were against the sutured pleura. The two flaps of periosteum were then sutured over the hernial orifice with No. 1 chromic catgut. The pleura was closed with No. 0 plain catgut, overlying muscle with No. 1 plain catgut and the skin with black silk. No drainage.

Clinical Course.—The period of hospitalization was 30 days. The patient showed a postoperative rise in temperature to 102° with pulse from 112 to 120. This subsided somewhat and rose again to 102° on the fourth and fifth postoperative days, then gradually subsided to a level of about 1° above normal at which it continued for about two weeks. Pulse and respiration were in proportion. The patient expectorated thick greenish sputum, amounting to about 8 ounces in 24 hours. This was probably due to his bronchiectasis. His general condition was good. On the seventh postoperative day the skin sutures were removed. The wound was clean. On coughing a moderate bulge was perceptible over the site of the hernia.

X-ray examination of the chest five days after operation showed "positive radiographic evidence of a lung collapse on the right side to about one-half its total volume. From the level of the eighth intercostal space down there is positive radiographic evidence of fluid." X-ray examination two days later showed "the fluid level is still present and the shadow at the site of fluid seems to be thicker than at the last examination. The markings of the right lung are completely masked by the fluid."

Seven days after operation, thoracentesis was performed withdrawing some 300 c.c. of air and 330 c.c. of yellowish clear fluid. Eleven days after operation the 24 hour sputum production was only 2 ounces. Culture of the chest fluid after 48 hours was negative.

The patient was put on general ultra-violet radiation. Cough and expectoration continued to diminish markedly. The wound healed well and the patient was discharged one month after operation much improved with no movement whatever at the site of the hernia on straining.

Hernias of the lung are classified on the basis of location and etiology. According to location (1) diaphragmatic (2) thoracic (3) cervical. According to etiology the hernias may be classified into two great groups, congenital and acquired. Acquired hernias are of 3 types; traumatic, spontaneous and pathologic. As examples of these, mention might be made of first, hernia caused by external wounds or following a fracture; second, congenital defect of the chest wall; third, a hernia resulting from coughing or a long strain.

The physiology of normal respiration is such that during inspiration and expiration there is never any tendency for the lung to produce hernial protrusions, because the lung follows the excursions of the chest wall and diaphragm. Under certain conditions the intrathoracic pressure is raised, such as by coughing or by the playing of wind instruments. Where there is a place of lowered resistance, be it congenital or acquired, this increased pressure will act against this weakened area of the chest wall and a hernia may follow. Of the three sites of occurrence of lung hernia above mentioned, the diaphragmatic is the least common. Beale's case is thus far the only reported diaphragmatic lung hernia. This is probably due to the fact that the intraabdominal pressure and the negative intrathoracic pressure offer such good buffer support to the diaphragm. Hernia of the superior aperture of the thorax is next most common and illustrates a place of lowered resistance. The factors controlling the protrusion of the lung in the cervical area are the parietal pleura, the cervical fascia and the overlying neck muscles. The usual site of herniation in this area is between the scalenus anticus and the sternomastoid muscles. The intercostal type of

hernia is the most common and usually occurs near the sternum and vertebrae, because the external intercostal muscles extend forward only as far as the costochondral junction and, behind, the internal intercostal muscles extend only to the rib angle. Most intercostal hernias occur anteriorly, because the pectoral muscles cannot give as good support as do the large spinal muscles posteriorly. Congenital hernias may occur in any of the three above-mentioned areas, and especially near the sternum. Hernias vary in size up to the dimensions of an orange. True hernias may be single or multiple, unilateral or bilateral and usually have a pleural sac. De Bruin has reported a case of congenital bilateral hernia of the pulmonary apices which extended into the neck in a female infant six and one-half months old.

Auler and Urbach reported the relative frequencies of the various types as congenital, 24; traumatic, 47; and spontaneously acquired, 31 per cent. Lutz and Montgomery from 165 cases reported congenital, 18; traumatic, 50; and "spontaneous," 32 per cent. In the analysis of cases to date the percentage of traumatic cases is slightly increased. According to Lutz and Montgomery from a series of 78 cases, the locations were as follows: cervical, 16, of which ten were on the right side and six on the left side; anterior chest wall, 57 cases, 34 on the right and 23 on the left; while only 5 were found on the posterior chest wall, 3 on the right and 2 on the left. Pulmonary hernia occurs much more frequently in males than in females. The age incidence is difficult to estimate for any one type of hernia, because of the above mentioned difficulty of distinguishing late traumatic cases from unrecognized late congenital ones.

It is sometimes difficult to classify a lung hernia on the basis of congenital or acquired etiology. A hernia which has for its basis anomalous development of the chest wall due to intrauterine bands, lack of fluid, etc., offers no difficulty in classification. As a rule fibrous elements replace congenital abnormalities in bony defects or arrested development. Due to the fact that the lung does not attain its complete expansion in infancy, we can understand why some of these hernias manifest themselves only in later life. On the other hand, traumatic hernia in early childhood may be overlooked and that hernia subsequently classified as congenital. Perhaps Hochsinger's suggestion might be well taken that all hernias occurring in the first few weeks of life must be considered as congenital.

Traumatic hernias may be divided into main groups, the first or false traumatic. These hernias consist of lung tissue emerging through the thorax in consequence of a wound but without a hernial sac. The second or true traumatic hernias are those which possess a sac composed of parietal pleura. These hernias may appear immediately or at a later time after the injury. The immediate cases usually supervene as a continuation of the traumatism, while the delayed ones appear in the course of the cicatricial repair process and its subsequent weakening when the lung exerts a continuous pressure against the chest wall. For instance, postoperative her-

nias of the chest wall may be produced by drainage tubes; for example, in empyema. Morel-Lavallee's division of traumatic hernia has a subdivision of consecutive hernia. This subdivision makes the classification unnecessarily complicated. This type is illustrated by postoperative hernias of the chest wall mentioned above.

Penetrating wounds of the chest wall are important factors, and yet it is most surprising to learn that during the American Civil War only three cases were reported, and during the Russo-Japanese War, among at least 20,000 wounds of the chest, only five cases occurred. Statistics of the number of hernias of the lung observed in the American Expeditionary Force are not available.

The rarity of protrusion of the lung in such cases is due to the inherent elasticity of the lung which causes it to contract and recede from the chest wall when the intrapleural tension reaches atmospheric pressure. Perhaps a better term for this type of hernia would be traumatic prolapse. Very often these cases are accompanied by pneumothorax and hemothorax. True hernias of the lung have rarely followed thoracoplastic operations if subperiosteal resections of the ribs are carefully carried out.

Spontaneous hernias are the result of intrathoracic disease, such as pulmonary tuberculosis, bronchitis and bronchiectasis resulting in coughing and straining. Diseases of the chest wall may also produce this type of hernia, such as caries of ribs, empyema necessitatis, malignant disease, etc. Finally this type of hernia may be the result of long-continued increased intrapulmonary pressure as in the case of musicians playing wind instruments, etc. Urbach in a study of 26 cases of spontaneous lung hernia found the following distribution: supraclavicular, 7; first interspace, 6; second interspace, 2; fourth interspace, 1; fifth interspace, 2; sixth interspace, 3; seventh interspace, 2; eighth interspace, 1; and sternum, 1.

The cardinal symptoms are (1) bulge (2) cough (3) pain at the site of the bulge. The symptoms may vary somewhat in accordance with the location of the hernia and to some extent with the type of hernia. The onset is usually insidious and is usually accompanied by pain and cough. If there is no underlying pulmonary disease, the cough will be nonproductive. It is chronic and spasmodic. The expectoration may be bloody. The bulge only makes its appearance during strong expectoration or coughing and disappears almost as soon as the intrathoracic pressure has abated. However, hernias of the lung, as in other parts of the body, may become incarcerated and strangulate, such as the cases reported by Koennecke, Verdeau-Caducee and J. Desse.

Physical examination reveals no abnormalities as a rule; and as the patient coughs a bulge suddenly makes its appearance. The size and the shape of this protrusion will be dependent upon the size of the aperture in the chest wall. Bony deformity or a scar may be at the site of herniation. On palpation, the neck of the sac may be felt. This may be a bony or fibrous ring through which a finger may readily be pushed into the chest cavity.

On forced expiration the lung can usually be felt between the fingers in the bulge, giving a soft crepitant sensation. Tactile fremitus is increased, the percussion note is resonant and tympanitic. The auscultatory findings are dependent upon the underlying pathologic condition. Vesicular breathing and crackling râles may be heard as the lung is being held between the fingers. The diagnosis of this rare condition should occasion no difficulty if the examiner will keep the possibility of its existence in mind.

A careful history and a thorough examination of the entire chest, particularly the chest wall, and the characteristics of the bulge will make the diagnosis readily. However, it will sometimes require diagnostic acumen to differentiate localized subcutaneous emphysema, perforating empyema, aneurysm of the intercostal vessels or aorta, fibroma, lipoma, and hemangioma of the chest wall from hernia of the lung.* Fluoroscopy and x-ray of the chest wall will readily establish the diagnosis in most of the obscure cases. Complications are rare. Strangulation resulting in gangrene of the lung has been mentioned above as has also hemoptysis.

Hernias of the lung rarely heal spontaneously. Per se they are rarely fatal except with the onset of complications. The treatment is essentially surgical. Radical treatment is strongly to be preferred, even though there have been some cases reported which healed themselves. These cases, however, became strangulated and the hernial orifice subsequently closed. The type of treatment must be based upon the underlying pathological factors and the amount of cough. The occupation of the patient sometimes warrants the greatest consideration. Palliative measures consist of rest, change of occupation and local compression of the hernia with pads and elastic devices. In the presence of cough radical operation should be delayed until the cause has been determined, and appropriate treatment to lessen the cough has been begun. When strangulation has developed, radical surgery is, of course, immediately indicated. The simplest surgical procedure in these cases is extirpation of the gangrenous portion. Great care must be exercised not to go beyond the neck of the sac. After the radical treatment in nongangrenous cases there should be maintained, when possible, a moderate pneumothorax for a short time. Temporary moderate pneumothorax is advocated, because the writer believes that the air will act as a cushion at the site of repair when this protection is most needed. There have been various types of plastic operation proposed, such as the transplantation of ribs, fascial repair, splitting of the rib adjacent to the hernial orifice and then using this bone flap as a covering for the hernial opening. Tuffier has reported the cure of a case by simply ligating the hernial sac. Graham has closed the hernial sac by repeated gauze packings. In some cases periosteal plastic closure of the hernial sac has given good results. The latter type of operation was used with excel-

*From personal communication with Dr. Howard Lilienthal, who related the history of a patient on whom he was operating for a lung hernia. The protrusion, though giving the usual signs of hernia, proved at operation to be a hemangioma of the chest wall.

lent result in the repair of the hernia reported in this paper. However, because individual experience has been so small, it is difficult to estimate the value of any one procedure, so that a standard method for the treatment of hernia of the lung cannot at the present time be recommended.

SUMMARY

A review of the literature of hernia of the lung, revealing to date 171 cases, is presented. Hernia of lung is a benign lesion and its recognition is easy if the possibility of its occurrence is kept in mind. Complications apparently are rare. The treatment is essentially surgical and no definite technical procedure can be outlined as being the best, because the individual experience has been so meager.

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THE MECHANICS OF ERUCTATION

A Hitherto Undescribed Function of the Diaphragm*

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ERUCTATION is a phenomenon which is commonly seen in different types of individuals both in health and in disease. It is a phenomenon that occurs primarily in individuals who swallow air under normal conditions (aerophagi), and also in those individuals in whom there is an obstruction in the pylorus, thus producing fermentation of the gastric contents with a resultant production of gas. It is considered a sign of good health among the Mohammedans, and belching to them is a sign of good digestion so that immediately afterwards they thank Allah for helping them digest their food properly. In some countries belching is considered bad etiquette and people make a voluntary effort to control such an act. The mere fact that eructations are easily controlled and can be produced at will, would lead one to assume that belching must have something to do with a voluntary muscle. In our observations we have found that the diaphragm is the muscle concerned, and its activity in the process of belching is to our knowledge the only activity in which one finds an independent and opposite action between the right and left leaf of the diaphragm. Under normal conditions during the respiratory contractions of the diaphragm both leaves go down during inspiration and go up with each expiration. Sometimes there may be a slight variation in height when one compares the right to the left half. We have a different state of affairs, however, when the diaphragm is used in the function of belching or eructation. In this case there is an independent and opposite activity between the right side and left side of the muscle.

Under normal conditions when a person takes a certain amount of food in the stomach, air usually finds its way into the stomach and this air, because of its lower specific gravity as compared with the gastric fluids, naturally finds its way up towards the fundus. In a study of the relation of the stomach to the hiatus esophageus of the diaphragm[1] we have found that the position of the stomach is such that it lies underneath the diaphragm in such a way that there is a definite kink at the cardia. This kink produces a definite closure of the gastroesophageal junction, and the lumen of this tube opens with each expiratory relaxation of the diaphragm and closes with each inspiratory contraction of the diaphragm. This opening and closure at the cardia is due to the action of the diaphragmatic

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pillars which contract and relax coincidentally with the parietal portion of the diaphragm. On inserting the finger into the cardia through a gastrostomy opening, it is possible to feel a milking down contraction that is present coincidentally with the inspiratory contraction of the diaphragm. This is followed by relaxation at the cardia which is coincident with the expiratory relaxation of the rest of the diaphragm. The pillars of the diaphragm, therefore, play a definite rôle in the opening and closure of the cardia. We have also noted that the parietal portion exerts a definite influence in the regulation of the motility of the stomach.[2] In a fluoroscopic and x-ray film study of a series of different types of patients who presented no gastrointestinal pathology we have found that the diaphragm presses the fundus downward during its inspiratory contraction. At this moment the pars pylorica of the stomach dilates so that it is ballooned up at the time when the fundus is contracted downward and laterally. With ascent of the diaphragm the fundus goes up with it and expands so that the latter balloons up while at the same time the pars pylorica of the stomach appears to be more contracted. We have noticed that after a series of these intermittent contractions and dilatations of the fundus and pars pylorica of the stomach a contraction wave of a typical peristaltic type begins to appear at the incisura angularis. In spite of the definite knowledge of the various factors which have a definite effect in the contractility of the stomach, our observations lead us to believe that the diaphragm has a definite influence on the motility of the stomach by changing the intragastric tension and also by producing action currents in the musculature of the stomach so as eventually to induce characteristic peristaltic waves. This activity of the parietal portion of the stomach has been followed through in cases of pathologic paralysis of the diaphragm induced by phrenic nerve resections. In a paper which we presented at the Radiological Society of North America on December 8, 1931,[3] we stated our finding that the results induced by the paralysis of the diaphragm may be classified under three general headings, namely:

1. Patients in whom respiration is of the costal type.
2. Patients with left phrenic neurectomy.
3. Patients with right phrenic neurectomy.

We have found that in the patients with costal respirations the phrenic neurectomy induced little or no rise of the diaphragm after operation. There was only a minimal contraction of the healthy half of the diaphragm and only a proportionately minimal inspiratory rise of the paralyzed half even at forced inspiration. In such a type of patient we saw no changes at all in the motility of the stomach.

In the second group of patients with left phrenic neurectomy we have noticed a paradoxical inspiratory rise of the left diaphragm. The tone of the pillars was fair and the barium meal remained at the cardia a few sec-

onds during each inspiration. The motility of the fundus was minimal. The fundus remained ballooned up with a bubble of air. Eructation was difficult or even impossible in these patients. The pars pylorica was noticed to swing laterally and obliquely so that the movements of this portion of the stomach reminded one of the movements of the pendulum of a clock with the fundus acting as the stationary point. The degree of the altered motility of the stomach depended upon the tone of the right diaphragm. In addition to these lateral movements one also noticed a constriction at the pars pylorica with a definite dilatation of the parts immediately above it. The constriction progressed along a U-shaped course with one tip at the pylorus and the other at the incisura.

In the third group of patients in whom the right diaphragm was paralyzed there was only a slight deviation from the normal motility of the stomach. The tone in the cardia was normal and the fundus showed its normal ascensus and descensus coincident with diaphragmatic contraction and relaxation. The pars pylorica showed its relative contraction and dilatation inversely as the contraction and dilatation of the fundus. The right diaphragm being now higher than the left, it caused a more obtuse angulation at the cardia so that during an attack of coughing the cardia was opened up and caused a regurgitation of the gastric contents. This phenomenon explains why a large number of these patients complain of a tendency of vomiting during attacks of coughing. This is in contradistinction to the type of patient in whom the left diaphragm remained more or less stationary by its paralysis and in whom the angle at the cardia became more acute, thus causing the difficulty in belching and the tendency to gastric distress by the resultant air in the fundus which was unable to find its way out as it would under normal conditions.

These normal and pathologic changes occurring in the motility of the stomach as a result of contraction of various parts of the diaphragm, particularly a tendency to distress in left phrenic nerve resections, have convinced us that there may be some definite connection between the diaphragm and the act of belching and regurgitation. In a series of 100 consecutive cases that we have followed by means of a fluoroscope we have found that as the air reaches the fundus it balloons the latter and causes a definite kinking of the cardia. When the patient is asked to belch we noticed that the left hemidiaphragm contracts rapidly and intermittently several times and at this moment the right leaf relaxes itself and rises proportionately. This intermittent contraction on the left side with a coincident relaxation of the right leaf has a tendency to straighten out the acute angle at the cardia sufficiently to prevent a valve formation caused by the acute angulation. With a slight pressure on the abdominal muscles exerted by the patient, the air very promptly finds its way out into the esophagus and upward into the mouth. Some patients who are not in the habit of belching have considerable difficulty in producing this series of contractions and

relaxations and frequently we notice that these patients showed a coincident contraction of both the right and the left leaf. After repeated attempts at deep breathing many of these patients showed the characteristic diaphragmatic movements and the air found its way into the esophagus.

In left phrenic neurectomies where the diaphragm has been paralyzed, these movements are impossible to obtain with the result that the patients have a great deal of distress when air or gas finds its way into the fundus. Moreover, on account of the phrenic neurectomy the tendency of the diaphragm to rise higher than its normal position increases the acuteness in the angle of the cardia so that the upper reflection of the cardia acts as a valve and further prevents the escape of gas from the stomach.

CONCLUSIONS

The act of eructation undoubtedly depends upon the contractions of the diaphragm. A thorough search in the literature has convinced us that this phenomenon has never been previously described. The contraction of the left hemidiaphragm coincides with the rise of the right half. There is an independent and inverse activity of the two leaves of the diaphragm, in that one side contracts while the other side relaxes. The normal position of the cardia shows a definite angulation and this angulation must be eliminated before air or gastric contents may be squeezed into the esophagus. In paralysis of the left hemidiaphragm, because of the increased rise of the dome, there is an increase in the acuteness of the angle at the cardia. In addition to this anatomical change the functional effects produced by the paralysis of the muscle prevent the normal characteristic contractions which are seen in the act of belching with the result that these patients have a tendency to distress and inability to regurgitate.

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ETIOLOGY, PREVENTION AND TREATMENT OF POSTOPERATIVE HEMORESPIRATORY COMPLICATIONS IN THE SURGICAL TREATMENT OF TUBERCULOSIS. ENDOTRACHEAL ANESTHESIA COMBINED WITH BRONCHIAL SUCTION. (84 CASES—152 OPERATIONS)*

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I. INTRODUCTION

IN DEALING with postoperative complications in the surgical treatment of pulmonary tuberculosis, I shall consider exclusively complications occurring after thoracoplasties. Pneumothorax, phrenicectomies, multiple intercostal neurectomies or apicolysis do not give rise, as a rule, to cardiopulmonary complications of importance as long as the compression exerted by them is well regulated; at any rate, if they do, their etiology is the same as in thoracoplasty.

Thoracoplastic collapse of the lung in tuberculosis is certainly, with pneumothorax, the most efficient measure we possess today for the surgical treatment of this disease. Although it is no longer accompained by the tremendous mortality of 75 per cent as in the time of Brauer and Friderich, it still presents an immediate mortality of over 10 per cent, and a delayed mortality of 20 per cent. Alexander in 1925 gave a mortality of 13 per cent, Bull (in 169 cases) 10 per cent, Archibald 5 per cent in good surgical risks but 40 per cent in bad risks. Gravensen gave 10 per cent, Key 9.5 per cent, and Sauerbruch 5 per cent, but in the second addition of his treatise he reported 29 deaths out of 233 operations; that is, 13 per cent. In 43 cases with questionable indications only 14 per cent were cured and 14 per cent died (Carter). Berard and Dumarest lately gave a mortality of 14.2 per cent.

The great majority of these deaths, 83 per cent according to Strockling, and almost all of them according to Sauerbruch are due to pulmonary and cardiac complications. Shock, heart failure and aspiration or tuberculous pneumonia are reported as the principal causes of death. Massive absorption of toxins from the collapsed lung, mediastinal flutter, infection of the wound, postoperative hemorrhage and embolism do not exceed 10 per cent of all causes of death.

Besides the mortality we must consider the postoperative morbidity.

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There is no doubt that spread of the disease generally follows postoperative pulmonary complications. It is common experience that patients who have survived such complications respond less well than those who had an uneventful postoperative course and that a number of the former subsequently present spread of tuberculosis in the sound lung.

During the past two years I have endeavored to study more closely, both clinically and experimentally the causes of death and the methods of prevention and treatment of these postoperative complications. This study was carried out in our tuberculous center composed of Seaview Hospital and the tuberculosis division at Metropolitan Hospital of the City of New York (2000 beds). This problem was approached with the underlying thought of disclosing the fundamental physiologic factors rather than of mere palliation of clinical manifestations.

II. PATHOLOGIC PHYSIOLOGY

Our present knowledge of the etiology of postoperative complications in general and following thoracoplasties more especially is far from being crystallized. Numerous are the theories advanced on the production of *shock, heart failure* and the so-called *"aspiration and tuberculous pneumonias";* naturally, various and contradictory are the proposed methods for their prevention and treatment.

In a recent paper, Eppinger (1932), studying the "postoperative symptom-complex" with the cooperation of the surgical division of his hospital in Köln, (Germany) concurred in the opinion that the principal factor in the production of postoperative shock was paralysis of the peripheral vessels, arterioles and capillaries. The characteristic symptoms of shock, small, rapid and often dicrotic pulse, low blood pressure, generalized pallor with often a slight cyanotic hue, cold sweats, rapid shallow breathing and collapsed veins, often rendering impossible intravenous injections, are produced according to this author by "peripheral circulatory failure." Cannon and Bayliss (1919) and more recently Eppinger, called attention to the similarities between postoperative shock and the condition created by intravenous injections of peptone and histamine. It was thus interesting to investigate whether postoperative shock was due to the passage into the circulating blood of depressive substances generated in the operative area by the breaking down of the proteins of the traumatized tissues. Histamine or peptone intravenous injections in the dog produce a typical shock with the same paralysis of the peripheral vessels, peripheral blood stasis, reduction of venous return to the heart, decrease of the amount of circulating blood and fall of blood pressure. The clinical syndrome of postoperative nonhemorrhagic shock is perfectly reproduced by these injections; i. e., rapid, small pulse, cold sweats, shallow rapid breathing, decreased muscular tonus and collapse of the peripheral veins. Table I gives the decrease of minute blood volume and of circulating blood after intravenous solution of peptone in the dog (0.2 per kg., body weight).

TABLE I

SHOCK PRODUCED IN THE DOG AFTER INTRAVENOUS INJECTION OF PEPTONE (EPPINGER)

EXPER.	ARTERIAL BLOOD PRESSURE MM. HG.	VENOUS BLOOD PRESSURE CM. H_2O	MINUTE VOLUME C.C.	VOLUME CIRCULATING BLOOD C.C.
I. Before shock	175	12	1070	725
During shock	70	3	380	318
II. Before shock	185	11	1806	971
During shock	90	2	460	270

According to the above mentioned authors postoperative shock in the human would be of toxic origin and due to absorption of histamine in the operative area.

There are many objections to this conception. Blalock and his collaborators (1930-31) have shown that the drop in blood pressure is due not to the absorption of toxic substances but to the loss of plasma and blood in the traumatized area, and that it was proportionate to the loss of fluids from the circulating blood. In dogs the femoral artery was dissected free and the thigh was constricted proximally with a tourniquet after the head of the femur was resected, so that all venous circulation was intercepted; the artery was left outside of the tourniquet. The muscles of the leg were then crushed distally to the ligature; shock was produced exactly as in animals without ligature of the member and often by a less severe traumatism. Guttentag has never been able to find histamine in the blood after traumatic or postoperative shock. Franken and Schurmeier have shown that shock can be produced by inhalation anesthesia without any traumatism or operation. This condition should also be distinguished from heart failure (Eppinger); in the first there is a marked decrease of the volume of the circulating blood because of the marked peripheral stasis which reduces the amount of venous blood returning to the heart, and causes incomplete filling of the heart and collapse of the veins. The size of the heart itself therefore is smaller, which is in contrast to the hypertrophied or dilated heart of cardiac decompensation and failure. Furthermore, in heart failure there is neither decrease in the volume of the circulating blood, nor decrease in the amount of blood returning to the heart.

On the other hand, it seems difficult to accept the toxic origin of postoperative shock in thoracoplasties. In fact, in the well regulated operation although traumatism is almost the same in cases operated upon by the same surgeon, the operative shock is most variable. Besides patients exhibiting the most alarming symptoms of shock, there are others presenting no symptoms and having an uneventful postoperative course. For these reasons I think that we should discard the toxic theory as it does not offer a satisfactory solution to our problem.

It is not the same with the theory of anoxemia and acapnia, the latter advanced by Henderson and his collaborators. According to Henderson and

Haggard, shock is due to oxygen and carbon dioxide deficiency. Anoxemia produced by insufficient pulmonary ventilation, whatever might be its cause, brings about rapid shallow breathing, the result of which is the washing out of the lungs of the extremely diffusible CO_2 (25 times more diffusible than oxygen). Decrease of the partial tension of CO_2 in the blood will cause increased oxygen deficiency in the tissues because of the "Bohr effect." In fact, Bohr, Hasselbach and Krogh (1908) have shown that when the partial tensions of CO_2 vary the blood takes up different percentages of O_2 although exposed to the same partial tension of the latter gas. The greater the partial tension of CO_2 the more the curve of dissociation of oxyhemoglobin is shifted to the right, that is, it more readily gives up oxygen. On the contrary, the lower the partial tension of CO_2 the

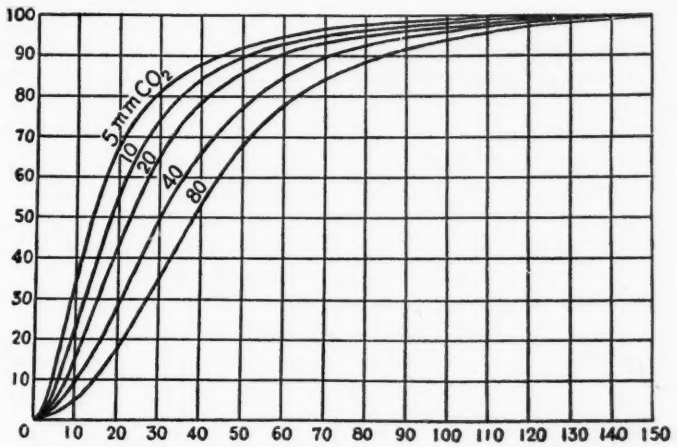

Fig. 1.—Curves representing the percentage saturation of hemoglobin with oxygen at different partial pressures of oxygen and CO_2. Dog's blood at 38° C. Ordinates = percentage saturation with oxygen; abscissae = partial pressures of oxygen in millimeters of mercury. (Bohr, Hasselbach, and Krogh.)

more the curve shifts to the left, that is to say, the more the hemoglobin holds on to its CO_2. Furthermore, as the CO_2 decreases, the hemoglobin is saturated with O_2 at lower levels of this gas. This phenomenon explains the fact that as CO_2 decreases, cyanosis becomes less apparent although oxygen deficiency remains the same (Fig. 1), since cyanosis depends exclusively on the percentage of reduced hemoglobin. This point is of great clinical importance. As Haldane, Meakins and Davies and others have pointed out, acute anoxemia with acapnia may pass unheeded both by the patient and the physician because of the lack of subjective symptoms. Contrary to asphyxia with retention of CO_2, as in obstruction of the respiratory ways, in anoxemia with acapnia there is little or no cyanosis, no respiratory distress, no gasping for air; the patient has a "pleasurable feeling of tingling narcosis" (Meakins and Davies), and death can occur

without struggle. On the other hand excessive elimination of CO_2 produces loss of tonus of the muscular tissues throughout the organism (Henderson). Skeletal muscles as well as vascular musculature present an excessive degree of relaxation. The arms and legs become limp, the diaphragm rises and moves very little, if any, during respiration, the respiratory muscles lose their tonus so that the thorax is in a position of deep expiration. The direct results of this condition are: decrease of vital capacity, deficiency in pulmonary ventilation and drop in the blood pressure. Dale and Evans have shown that the latter is not due to any direct influence of acapnia upon the heart but to the depression of the normal activity of the vasomotor nerves in the bulb and the spinal cord. Starling believes that "CO_2 has a double effect on the blood vessels: a central effect through the vasomotor centers of the medulla and spinal cord, causing contraction of the blood vessels; a peripheral effect, causing dilatation of the blood vessels. The general effect therefore will be to cause dilatation of the blood vessels on the part where the carbon dioxide is produced and where it is present in greatest concentration and vascular constriction elsewhere under the influence of the nervous centers."

Whatever it may be, muscular tonus is of the greatest importance because it enables us to understand the limitation of respiratory movements as was pointed out by Churchill and McNeil, Powers, Sise, Head and others. Reduction of the vital capacity during the first days following operations may be two-thirds normal and develops even in the absence of pain or splinting of the chest. More recently Muller, Overholt and Pendegrass have emphasized postoperative elevation of the diaphragm and the narrowing of the intercostal spaces. A status of hypoventilation is thus created which may last several days after operation. Eppinger followed the variations of the perimeter on the chest and of the vital capacity in a man of 44 operated upon for gastric ulcer. His findings are given in Table II.

TABLE II

POSTOPERATIVE CHANGES IN THE CHEST PERIMETER AND THE VITAL CAPACITY (EPPINGER)

	BEFORE OPERATION	AFTER OPERATION				
		½ hr.	1 day	5 days	9 days	12 days
Thoracic perimeter	7	4½	3	5	6	7
Vital capacity	3,100	2,000	1,500	2,150	2,600	3,100

It is evident that this partial functional collapse of the lung will have three effects; first, inadequate ventilation and, consequently, creation of unaerated channels and insufficient oxygenation of the circulating blood. Second, impaired bronchial drainage because of decreased pulmonary ventilation and lowered expulsive efficiency of cough; third, as a corollary of bronchial stasis, of impaired drainage and of narrowing of the diameter

of the bronchi (expiratory bronchial collapse), bronchial obstruction and production of atelectasis or pneumonia becomes easier. In fact the position of the lung in these cases, the "acapnial position" says Henderson "may be described as one near or below that of normal expiration, a condition of prolonged deflation. As long as this position is maintained, the thoracic, diaphragmatic and abdominal movements of breathing are carried on at a shallow level. Inspiration inflates the lungs only slightly and expiration deflates them almost as completely as the deepest voluntary expiration. This deflation leaves some parts of the lungs unventilated, thereby permitting an accumulation of mucus, a blocking of airways and the production of atelectasis."

Collapse, and even more so atelectasis or pneumonia, produce, as Birnbaum and I have shown, suppression of ventilation in the affected areas long before suppression of circulation. This explains the creation of unaerated channels in the lung which prevent adequate oxygenation of the venous blood circulating in these areas, so that this inadequately oxygenated blood is mixed with and pollutes the blood which returns to the left heart from well aerated pulmonary areas and is consequently, adequately oxygenated. Another fact of importance, which has never been pointed out, so far as I know, is that thoracoplastic collapse itself of the diseased lung is conducive to the creation of unaerated channels, further increasing the unaerated pulmonary field. These facts give us a clear explanation of the discrepancy between respiratory and circulatory capacity of the affected areas. As Green has pointed out, if the alveolar capacity is decreased to 60 per cent of normal the pulmonary blood volume will be practically normal but the aeration only 60 per cent efficient. It is obvious that such blood has an oxygen content not very different from 60 per cent of the normal oxygen capacity. With normal venous blood which is already from 50 to 70 per cent saturated with oxygen, a 60 per cent deficiency in aeration will produce no great damage. But when the venous blood comes to the lung largely unsaturated, because of peripheral stasis, then 30 per cent deficiency in oxygenation may cause a two-thirds unsaturation which is the critical maximal percentage that maintains life. Barrach has stressed the same point in lobar pneumonia (1922). A vicious circle is thus created. Oxygen unsaturation of the blood affects the respiratory center and causes rapid breathing, which impairs ventilation by increasing the pulmonary dead space (Haldane, Dautrebande). Rapid shallow breathing brings about washing out of carbon dioxide and further anoxemia to the tissues because of the Bohr effect, already mentioned. Thus anoxemia rapidly increases, leading to anoxemic crisis as will be shown below.

The whole question of shock following thoracoplasties seen from this angle becomes clearer and much less confused. More recently Henderson advanced a newer conception which throws more light on the problem. This is the "veno-pressor mechanism." According to this author "the circulation should be conceived not as a balance of two mechanical factors

only, the heart and the vasomotor system, but rather of three factors. It is supported like a tripod that falls if one of its three legs falls. The heart pumps the blood onward; the vasomotor nervous system regulates the caliber of the finer blood vessels and thus determines the arterial resistance and pressure. But it is chiefly a third factor, the 'veno-pressor mechanism,' that pushes the blood back through the veins toward the right side of the heart." Riml has shown that if the chest of a rabbit is opened and the pulmonary artery is clamped, the pressure in the vena cava rises, although the "push exerted by the arterial blood had been suppressed by the clamping of the pulmonary artery." According to Henderson the venous return is chiefly accomplished "by the general tonus of all the skeletal muscles of the body, more especially of the abdominal muscles and particularly the diaphragm as well as the nonstriated musculature of the alimentary canal." This muscular tonus is controlled by the respiration in its broadest sense, that is, the exchange of gases in the tissues, which are regulated by both respiration and circulation.

Under physical exertion, respiration, muscular tonus and circulation increase together as they decrease together in physical depression as after a major operation. Wiggers not only believes in the venopressor mechanism but also has shown experimentally that variations of CO_2 have no effect whatever on the heart itself when all other conditions were kept the same, i. e., the heart rate, the venous pressure and the arterial resistance. It is time to concede that in the so-called "weak heart action or heart failure" following operations or hemorrhages, the heart is not at fault; in reality it is the volume of the venous return to the right side of the heart and its oxygen content that are deficient (Henderson). Dale and Evans have shown that under anoxemia-acapnia conditions the inflow of the blood to the heart is decreased and the heart having only a small amount of blood to contract on looks "hypertonic," i. e., reduced in size. That this is so is proved by the fact that this condition may at once be removed by O_2- CO_2 (5 per cent) inhalations, which clearly differentiates this syndrome from cardiac decompensation with dilated heart.

In stressing the point of the paramount importance of oxygen-carbon dioxide want in the production of postoperative shock, we should not underestimate the importance of the previous condition of the cardiac muscle and of its pathologic changes which may considerably decrease its resistance. Neither should we overlook the fact that in collapse treatment for tuberculosis the more or less complete collapse of one lung is followed by a corresponding collapse of the capillary vessels of the same lung thus decreasing the area of the lesser circulation and correspondingly increasing the pressure in the right heart. H. Jessen (1932) in a thorough study of the venous pressure in pulmonary tuberculosis and therapeutic collapse of the lung has reported increased venous pressure in the fibrotic (atelectatic) forms of tuberculosis; in pneumothorax or thoracoplasties on the right side the same author found 50 per cent higher venous pressure

than in such operations on the left side, probably because the right heart is more easily compressed than the left heart; these findings explain the well known fact that right thoracoplasties have a more serious prognosis than thoracoplasties on the left side.

With these restrictions made, it is my contention that in the last analysis shock and early postoperative deaths following thoracoplasties are due to oxygen want which leads to the so-called "anoxemic crisis." It is of interest to further study this condition because upon it is based the prevention and curative treatment which I shall describe below.

Koehler and his associates believe that "anoxemic crisis" and death is due to failure of the supply of energy required to maintain the physiochemical relationship of the cell. The tissues which are affected first are the ones which metabolize more; it is therefore natural that nerve cells which present the highest metabolism (Parker) are affected by anoxemia earlier and in a more marked degree than the other tissues. Partial anoxemia will produce a functional disablement of nerve cells without touching the other tissues and this is the physiologic mechanism of general narcosis by inhalation anesthesia. Prolonged partial or acute complete anoxemia will produce disorganization and death of the cells of the respiratory and circulatory centers long before the other tissues are affected.

Direct action of anoxemia upon the heart and the blood is of no less importance. Clark, Engleton and Evans have shown (1931) that when perfused frog's heart is deprived of oxygen, glycogen very rapidly disappears, or, more exactly lactic acid and its precursor phosphatides cannot be oxidized again into glycogen. Furthermore, when the vagi are intact, the action of progressive anoxemia can be summarized as follows (J. Sands and Degraff). In progressive anoxemia, anoxemic crisis occurs when the percentage of oxygen has been reduced to about 9 per cent, (the minimum oxygen pressure required for continuation of life being around 30 mm. of Hg, equal to 7 c.c. of oxygen per 100 c.c. of blood). Below it the respiratory center is greatly affected. Previous to the crisis, when the vagi are intact, the heart rate increases progressively, the systolic pressure tends to rise, tending to increase the pulse pressure. This appears to be due to the combination of cardiac acceleration, reduced peripheral resistance and increase in systolic discharge. The latter is under the influence of two opposing effects of anoxemia upon the heart; on one hand reduction of the effective venous pressure and decrease of the fundamental filling pressure; on the other hand, rise of the initial tension in the left ventricle and increase of the rate of ventricular ejection. During the early stages of progressive anoxemia these two factors remain evenly balanced, and tend to maintain systolic discharge normal or to increase it slightly. Thus the minute flow of the blood through the body is increased during that phase of anoxemia under the combined influence of increased systolic discharge, increased heart rate and reduced peripheral resistance.

When the circulatory anoxemic crisis occurs, systolic and diastolic pres-

sure decline and pulse pressure falls. The minute output of the heart decreases whereas venous pressure rapidly rises. This picture is one of circulatory failure, which precedes the final slowing of the heart, and the cessation of respiration.

Up to this time oxygenation of the blood will rapidly relieve the condition. It is not the same when the heart slows down; tissue anoxemia is then very rapidly increased because of the rapidly diminishing minute cardiac discharges, and disorganization of the nervous centers rapidly follows. The accompanying curves (Figs. 2 and 3) illustrate the course of events during anoxemia. The first was taken during experimental submersion (in the dog); the second during respiration of a dog in an atmosphere of nitrogen.

The action of anoxemia upon the blood is also to be considered. According to Clark, Engleton and Evans it is diphasic. At the beginning

Fig. 2.—(Dog 625.) Submersion in tap water. Light ether anesthesia. Submersion (1) is immediately followed by rise of blood pressure (c.d.f.) of 1½ min. duration, and then gradual drop and stop of the heart (3 min.) Respiration shows a period of dyspnea followed by deep slow respirations and terminal apnea. Animal died. (Time in seconds and minutes.) The exact moment of taking blood specimen is marked.

alkalosis is produced due to the rapid washing out of CO_2 following hyperpnea and shallow rapid breathing. But soon progressive anoxemia of the tissues causes accumulation of fixed acids which compensate alkalosis; if anoxemia progresses the most extensive grade of acidosis develops, and a vicious circle begins; increased anoxemia shifts the P_H to the acid side, and increased acidosis further interferes with the power of the tissues to use O_2.

This rapid review of the physiologic pathology of respiration shows the close relation of respiration and circulation and their interdependence following disturbances of either of these two functions. The problem is neither simple nor of such minor importance as to deserve the little attention and thought that is given to it by surgeons and even by internists. It would be difficult to deny the fact that so far, little consideration has been

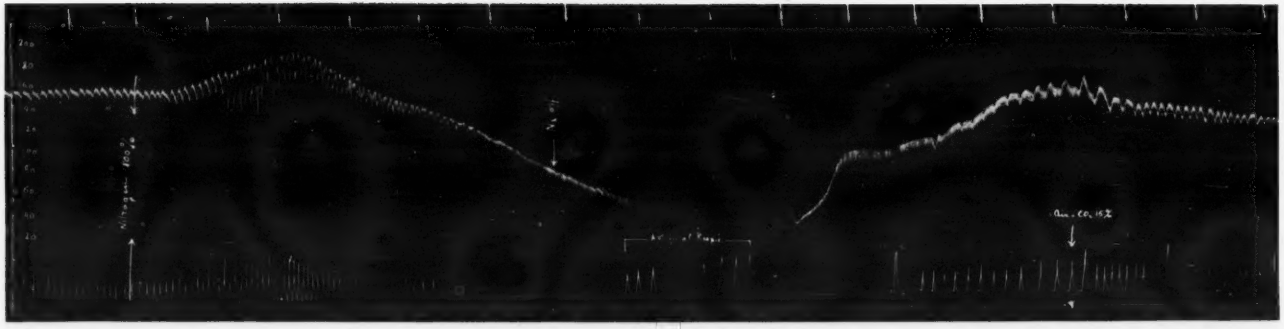

Fig. 3.—Resuscitation after asphyxia by breathing pure nitrogen. Absence of initial apnea. Respiration became deeper and more rapid. Two minutes after the beginning of the experiment apnea occurred. The blood pressure after a rise (1 min. ½) started dropping very gradually. Three min. after the beginning of the experiment administration of N_2 was suppressed, and a few intratracheal inhalations of air containing about 4 per cent of CO_2 (alveolar air of one of us) were given. After ½ min. blood pressure started rising again and spontaneous respiratory movements began. (Time in half minutes.)

given to the physiology of the lung in the study of its pathologic changes, and their clinical manifestations. Lack of specific knowledge has been the cause of the adoption of such generic, vague and unscientific terms as "congestion of the lung" (outside of cardiac decompensation), "heart failure" or "respiratory failure."

The development, however, of a progressive and aggressive thoracic surgery, the increasing importance of surgical treatment of chronic pulmonary suppurations and more especially of pulmonary tuberculosis made it necessary to revise many generally adopted opinions and theories on that subject. Thoracic surgeons have been faced with sudden and often puzzling catastrophies which could neither be explained with the vague notions so far advanced in clinical textbooks nor be fitted into the generally adopted patterns. A different approach to this problem was necessary. The careful study, both clinical and experimental, of the pathologic physiology of atelectasis, pneumonia and postoperative pulmonary complications carried on persistently by Birnbaum and myself and other investigators for a number of years, made possible this different approach and opened up new roads. A better knowledge of the mechanism of their production and of their close relation with the physiologic function of gas exchanges in the lung, has rendered possible the explanation of the interaction and interdependence of these complications with oxygen and carbon dioxide deficiency on one hand, and on the other, it led us to recognize the great importance of aerodynamic disturbances in the lung.

Thus the study of the so-called "aspiration or tuberculous pneumonias" following thoracoplasties should not begin at the point where this complication is established but long before this point is reached, that is, at the early stage of their inception. By paying more attention to the mechanism of their production than we have done so far, we may closely approach the solution of the problem of their prevention which, I believe, is the best way of treating them. It is along the lines of these general principles that the study of these complications are presented here.

III. HEMORESPIRATORY COMPLICATIONS FOLLOWING THORACOPLASTIES

Let us first understand that respiration does not take place in the lung, any more than does the circulation in the heart. The lung is the organ in which only ventilation of the blood takes place, by a very accurate and extremely sensitive coordination of the aeration and circulation in that organ. The real act of respiration takes place in the tissues, where oxygen is delivered and CO_2 carried away. As in the lung, so in the tissues, an extremely accurate coordination between gas exchanges and circulation is necessary for maintenance of normalcy. The respiratory needs of the tissues vary continuously; the amount of oxygen necessary and the amount of CO_2 produced change from second to second, causing corresponding changes in the acid-base equilibrium and the P_H of the tissues. The latter regulates the important functions of water metabolism and membrane

permeability. These minute adjustments, which require instantaneous and continuous changes in the amounts of blood brought to each individual tissue, in the duration of contact of the blood with them and in oxygen content of the blood, are accomplished by continuous and accurately corresponding changes in the size of the small vessels. This continuous play of arterioles and capillaries renders possible the maintenance of normal conditions in the tissues.

This shows the necessity of the closest cooperation of respiration and circulation and it explains the mechanism of the regulation of both by the needs of tissue metabolism. The algebraic sum of metabolic activities in the tissues of the organism regulate both pulmonary and cardiac activities. Haldane has expressed it by saying that "respiration and circulation are regulated by gas exchanges in the tissues." In other words gas exchanges in the tissues and changes in the peripheral vessels are the regulators of respiratory and circulatory functions. We can thus conclude that in the study of the pathologic changes of respiration and circulation, following thoracoplasty, we should consider together and as a whole the changes occurring both in respiration and in circulation, and this not only in the lungs and in the heart, but also in the innumerable and most important "peripheral lungs and hearts."

It now becomes easier to attack the problem of the production of postoperative hemorespiratory complications in thoracoplasties and their preventive and curative treatment.

The delivery of oxygen to the tissues and the elimination of CO_2 is possible only if both respiratory and circulatory systems function normally. For this the following requirements must be satisfied. First, patency of the upper respiratory ways and of the bronchial tree; second, anatomical and physiologic integrity of the respiratory membrane of the air sacs; third, normal function of the pulmonary circulation; fourth, adequate amount of hemoglobin in the blood and a normal blood capacity for carrying oxygen; fifth, sufficient volume of respiratory air and normal total and partial pressures of its components, and sixth, a normal condition of the respiratory, circulatory and vasomotor nerve centers.

Before studying the above requirements, it is necessary first to point out the unfavorable conditions for normal respiratory function under which our patients are placed during thoracoplasty for pulmonary tuberculosis. In fact, not only is one of their lungs deeply affected, but chronic bronchitis also is always present and the heart muscle is more or less damaged. Furthermore inspiration in the healthy lung is impaired because of the position in which these patients are placed on the operating table and because of internal drainage of the exudate flowing by gravity, during operation, from the diseased lung. Archibald, Brunn and Faulkner, Faulkner and Faulkner, and lately McCordock and Ballon have presented thorough studies of the latter factor. During resection of ribs and the following col-

lapse of the diseased lung more exudate will be pressed from it and will pass into the bronchi of the healthy lung or the noncollapsed portion of the diseased lung. Thus the respiratory field of the lung will decrease and unaerated channels, as we have seen, will be created interfering with adequate oxygenation of the blood; right then and there the vicious circle of anoxemia, which has been already studied in detail, will begin.

What will happen to the exudate drained into the healthy portion of the lung? It is common experience that these patients cannot cough and expectorate during the first 24 hours following thoracoplasty. This is not due only to the pain but also to anoxemia and acapnia following impaired ventilation. It is obvious that according to the degree of viscosity of this exudate small or large size bronchi can be obstructed, so that pulmonary lesions of lobular or lobar distribution may develop. It is also obvious that "wet" cases, expectorating more than 30 c.c. of sputum are more exposed to complications than "dry" cases. I wish to point out, however, that I have had patients expectorating less than 10 c.c. in 24 hours, from whom more exudate was aspirated during operation than from cases expectorating twice as much or more. This is due, I believe, to the presence of large cavities with retention of exudate in them (fluid level) which cannot be emptied by any means before collapse of the lung because of the narrowness and tortuosity of the bronchial outlets. Postural as well as bronchoscopic drainage are equally ineffective. I have been repeatedly unable to aspirate any exudate before operation whereas during operation and especially after the upper two ribs were resected, from 4 to 10 c.c. of viscid exudate were aspirated. For that reason I do not think that Graham would have been more successful in avoiding postoperative pneumonia in his cases reported by McCordock and Ballon even if he had performed preoperative bronchoscopic drainage, or that Archibald and Dean Cole could have prevented this complication with postural drainage alone. I venture this opinion from a personal experience based upon 152 thoracoplastic operations for tuberculosis and bronchiectasis in each of which bronchial aspiration was performed before, during and after operation.

It is also not necessary that the sputum retained in the healthy portion of the lung be of considerable amount. The irritation produced by its presence will induce bronchial secretion from the mucosa of the healthy bronchi, since it is known that this is the mode of defense of bronchial mucosa to any mechanical or chemical irritation. This retention is sufficient to start the ball rolling; expiratory narrowing of the bronchi, due to anoxemia and acapnia, will render bronchial or bronchiolar obstruction easier. Another factor of importance is the loss of tonus of the bronchial muscles and the impairment of the peristaltic movements of the bronchi; I believe that these movements do exist and that they constitute an important means of defense of the lung.

Another point which I have investigated was the part played by the

pneumococcus in the production of these pulmonary complications. Routine examination, in 35 cases, for pneumococcus, first of the sputum of these patients before operation, second of the bronchial exudate aspirated from the bronchi before, during and immediately after operation, and third of the sputum expectorated during the three days following operation, gave us the following results.

In 32 per cent pneumococcus Group IV was found in small numbers in the sputa examined before operation; in 40 per cent of bronchial exudates aspirated before, during or at the end of the operation; in 100 per cent of cases in which pulmonary complications developed. Of equally great importance is the fact that in the exudate sucked from the throat and the pharynx at the end of the operation, pneumococcus was present, in 64 per cent of these cases. The appearance of numerous pneumococci in the sputum of the postoperative cases who presented some kind of pulmonary complication has been so consistent that this examination is carried on routinely. Appearance of numerous pneumococci Group IV in the postoperative sputum means impending complication and even in the absence of clinical signs, precautions and preventive measures are carried out, the most important of which is O_2-CO_2 (10 per cent) inhalations.

In conjunction with the above findings it is interesting to report the work of Gomez who was so much impressed by the constant presence of pneumococci in the sputum in postoperative pulmonary complications following thoracoplasty and even phrenicectomy that he proposed to immunize patients before operation by injections with polyvalent pneumococcus vaccine. Verdina repeatedly found virulent pneumococci in great numbers in the sputum of patients in whom hemoptysis was closely followed by a spread of tuberculosis. On the other hand in postoperative pulmonary complications following operations upon the abdomen, Whipple constantly found that pneumococci were present; these were of the same type as the pneumococci isolated from the throat of the same patients before operation. As Birnbaum and I have already pointed out elsewhere, the frequency of occurrence of these complications in patients operated upon while having a common cold can be explained by the work of Dochez and his collaborators. These authors have shown that in the human and more especially in the ape, while before experimental production of a common cold very few colonies of pneumococcus were present in plate cultures made from the sputum, after the inoculation of cold virus a tremendous increase of the organisms was observed as a rule.

The question which naturally arises is whether the mucous exudate of the throat can be aspirated into the trachea and bronchi during operation and thus contaminate the exudate retained in the bronchi because of the mechanical reasons already studied above.

Holscher (1898), in a remarkable paper, reported that if during ether anesthesia gentian violet was introduced into the mouth, it could be reg-

ularly found after a short while in the small bronchi. He came to the conclusion that most infections of the lung after ether anesthesia are not due to irritation produced by the ether but to aspiration of infected material coming from the mouth and throat. In a more recent work Lemon, experimenting with dogs under ether anesthesia found that when the animal was lying in a horizontal position or only slightly inclined, barium emulsion, lipiodol, silver iodide emulsion, india ink or gentian violet simply introduced in small amounts into the mouth could regularly be seen, after 40 minutes of narcosis, in the small bronchi. Of greater importance for us is the fact that broth cultures of bacillus prodigiosus introduced into the mouth of the animal under the same condition were found in cultures of the content of the small bronchi. Lemon and Higgins in their recent work (1932) came to the same conclusions. Only when the animal was placed in complete Trendelenburg position so that the feet were 28.75 cm. above the head could aspiration be partly prevented. Otherwise even heavy particles were aspirated by the inspiratory draft against gravity and against the movements of the cilia of the bronchial epithelium. The same phenomenon was ascertained by Myerson, who in bronchoscoping patients immediately after tonsillectomy, found that 90 per cent of all cases operated upon under slight general anesthesia and with laryngeal reflex retained, blood was found in the bronchi. Only in patients who coughed during operation was blood less often found in the bronchi (18 per cent). Vail (1928), Guisez (1930), and others reported similar results after bronchoscopic examinations.

Pneumococci and other organisms can thus be aspirated into the tracheobronchial tree during inhalation anesthesia. But even without anesthesia this is produced, and Harkavy has proved that the lung is not aseptic as it has generally been believed but that numbers of microbes are found as far as the third bronchial divisions.

These facts render easy, I believe, the formulations of the sequence of events leading to the production of postoperative pulmonary complications following thoracoplasties. The posture facilitates drainage by gravity of the material expressed from the collapsed lung into the bronchi of the healthy lung or of the noncollapsed portion of the diseased lung. As cough, expectoration, respiratory movements and to an unknown extent, idioperistaltic movements of the bronchi are suppressed or greatly diminished during the first 24 or 48 hours following operation, bronchial stasis may occur. This stasis will be of short or of long duration according to the degree and duration of the impairment of these means of defense of the lung. Exudate will be able to obstruct small or larger size bronchi and thus diminish the respiratory field of the lung, and, furthermore, create unaerated channels which further increase anoxemia. Two factors of importance in the further development of bronchial stasis are: the viscosity of the exudate and the nature and virulence of the microbes present

in it. In fact, moderately viscid exudate will be able to obstruct only small bronchi and produce patchy atelectasis and this only if previous lesions of the interlobular and interacinal septa have already suppressed collateral ventilation (Van Allen, et al.). On the contrary, very viscid exudate will be able to obstruct larger bronchi and cause lobar or even massive atelectasis. The viscosity of the exudate is increased when pneumococcus is present and its constant presence in all reported cases of postoperative atelectasis is certainly not a mere coincidence. Upon the virulence of the infecting pneumococcus and the duration of the impaired ventilation and free bronchial drainage will depend the further evolution of the complication. Birnbaum and I have presented experimental and clinical evidence to show that no clean cut distinction can be made between postoperative atelectasis and postoperative pneumonia, notwithstanding the contrary opinion lately advanced by Van Allen and Wu (1932). This is even more apparent following thoracoplasties. Lobular atelectasis and bronchopneumonias, or lobar atelectasis and lobar pneumonias are supposedly distinguished, when this is possible, only by the severity of the toxic symptoms which accompany them; otherwise physical signs, radiographic findings, bacteriologic and pathologic characters may present quantitative but never qualitative differences. The virulence, nature and number of microbes, the resistance of the patient and the duration of the obstruction are the only factors which determine the fate of the lung deprived of its means of defense.

The so-called "tuberculous pneumonias" and "tuberculous spreads" are produced I believe by the same mechanism. In a recent paper McCordock and Ballon, studying two cases of "tuberculous pneumonia" following thoracoplasty, in which autopsies were performed, found that the pneumonias of the good lung were produced by infected material which came from the diseased lung, so that "the bronchi contained plugs of material similar to that found in the tuberculous cavity which had been subjected to compression." The postoperative lesions could not represent "flare-ups" of already existing lesions in the good lung because such lesions were not found in the cases of these authors (especially in their case 1). "Large masses of tubercle bacilli," they say, "could be demonstrated in the smaller bronchi; the presence of tubercle bacilli could not be attributed to a preformed focus in this lung because there were no lesions older than the pneumonic process that could have supplied the bacilli necessary to induce pneumonia." The explanation of the production of these tuberculous pneumonias or bronchopneumonias in the good lung (following operations), although lesions failed to develop in it before the operation despite the presence of tubercle bacilli in the sputum, is given, I believe, by the impaired bronchial drainage. In fact, the importance of the number or "dosage" of bacilli in the development of bronchogenic pulmonary tuberculosis is well proved; as long as free bronchial

drainage is assured in the good lung the number of bacilli in contact with the bronchial mucosa remains relatively small; it is not the same when large amounts of tuberculous material are trapped in the bronchi. The good lung presents a degree of resistance which is finally overcome when the dosage of the infection reaches the strength required for this purpose. Animals naturally immune to tubercle bacillus can become tuberculous provided sufficient number of virulent bacilli be introduced into them (R. Koch), or when the resistance of the host has decreased, as when thyroparathyroidectomy is performed on the albino rat (M. Steinbach). It seems very probable that pulmonary infections as bronchopnuemonia, or influenza exert a similar action upon the resistance of the lung, rendering possible the development of tuberculosis in a lung which so far had resisted successfully. It is not within the scope of this paper to discuss the part played by tissue reactions of a hypersensitive and already allergic lung in the production of these postoperative complications of tuberculous nature. Tuberculin certainly is liberated by the tuberculous material which is poured into the healthy lung and forced by bronchial stasis and bronchial obstruction to remain there. I shall not enter the most delicate and greatly discussed premise that acute tuberculous infections may be exudative, (resolving within a few weeks or months without leaving any traces) or that may produce caseous necrosis of the parenchyma from the start (caseous pneumonic form of Ornstein, Ulmar, and Dittler). The production of these different forms depends upon dosage of infection. Therefore I cannot agree with Archibald, who in discussing the paper by McCordock and Ballon, said: "we find it (tuberculous pneumonia in the good lung) also occurring and clearing up inside of a month or two, which seems to me almost irreconcilable with acute tuberculous infection."

The fact remains clear that postoperative tuberculous spreads in the good lung are produced by tuberculous material expressed during operation from the diseased lung and allowed to remain in the good lung. If additional proof of this is necessary I can recall several similar spreads produced by the tuberculous sputum spontaneously or following a common cold producing edema of the bronchial mucosa and impairment of bronchial drainage as bronchial obstruction often occurs after hemoptysis or sometimes after lipiodol injections into the bronchi.

The consensus of opinion of internists and surgeons is unanimous in considering the tuberculous material expressed from the diseased lung as the principal factor of postoperative pulmonary complications.

Berry has reported (1929) four cases of massive atelectasis following thoracoplasties, with death in two cases, and 5 postoperative pneumonias in 93 cases which were operated upon on the service of Lambert and Miller. In discussing these cases he says: "there is no doubt in my mind that they are due to aspiration of mucous viscid exudate from the diseased lung." The same opinion is upheld by Alexander. "Postoperative pain in itself" says this author, "prevents normal coughing for days or even weeks, the

ribs are sectioned and prevented from moving properly and part of the force of cough is dissipated in bulging the 'de-ribbed' chest wall; many of the thoracic muscles are incised and crippled; it is obvious that the pain and crippling occasioned by operating upon the lower chest of one side prevents, to a large extent, proper coughing on both sides. This, together with the fact that the breathing lung of the unoperated side is more liable than the compressed lung to aspirate infected secretions, accounts for the higher incidence of pneumonia in the uncollapsed lung." Sauerbruch expresses the same opinion and therefore, he writes, "during the days following operation, every patient is encouraged by specially trained nurses to cough and expectorate."

Furthermore, statistics show that the healthy lung is more frequently affected than the diseased one, apparently the exudate flowing by gravity into that lung. In Brunner's series, pneumonia occurred 3 times in the collapsed lung and 5 times in the sound lung and twice in both. Strickling reported two pneumonias in the collapsed and four in the sound lung.

It is peculiar that whereas all authors agree in considering the exudate and its ensuing stasis as the determining etiologic factor, no serious effort has been made for its elimination. In fact, very little if anything has been done to eliminate the cause, while a great deal of discussion is being had over secondary factors, especially upon anesthesia to be used and the operative technic to be chosen in order to avoid these complications.

Sauerbruch uses local anesthesia in "wet" cases, but he prefers general narcosis when the patient expectorates less than 30 c.c. in 24 hours. Moreover, he considers that if local anesthesia is not complete, or if the patient is very emotional, so that restlessness and cough and deep inspirations are produced during the operation, it is preferable to resort to general anesthesia in order to avoid "aspiration into the small bronchi of the pulmonary exudate expressed from the collapsed and diseased lung during operation." Carter for the same reason uses local anesthesia with infiltration of the intercostal spaces. Davies uses chloroform, because, he says, ether increases bronchial secretion. Nissen uses general anesthesia when the patient expectorates more than 30 c.c. in 24 hours. Lilienthal, Archibald, Welles and Roux (of Lausanne) prefer general anesthesia, Picot(of Laussane) gave up local and uses general anesthesia, while Chalier, Sebrechts and others use rectal anesthesia. Magill combines oxygen-nitrous oxide with novocaine block of the intercostal nerves, and others advise avertine supplemented by general or local anesthesia (Charrier and Loubat). However, if one compares the statistics he will be surprised to find that the number of postoperative pulmonary complications remain essentially the same.

The same considerations apply to the various surgical technics advocated in order to prevent postoperative pulmonary complications. It is of real interest for this purpose to read the paper presented by Lilienthal at the annual meeting of the American Society for Thoracic Surgery

(1926) and more especially the discussion which followed. Lilienthal believes that the logical sequence of two stage thoracoplasty is to resect the upper ribs and in a second stage the lower ones; the arguments in favor of this technic are first that it allows the immediate collapse of the cavities generally situated in the upper lobe, thus suppressing the source of the infected material; second, that in a number of cases the upper stage alone suffices for bringing about clinical cure, without resection of the lower ribs. Archibald, on the contrary, follows the technic of Sauerbruch, that is, first resection of the lower ribs, because in this way he believes that "the lower lobe is collapsed and aspiration of exudate coming from the diseased lung is prevented." He admits, however, that the analysis of the results obtained by these two procedures does not solve the question in favor of the first or of the second. Alexander advises the collapse of the lower lobe by phrenicectomy and then thoracoplasty of the upper ribs. Welles followed the technic adopted by Lilienthal up to 1927; in discussing the above-mentioned paper of Lilienthal he said, "I believe that Lilienthal is right about the lack of danger of pushing secretions from the upper lobe cavities into the lower lobes because the lower ribs are the ones used for coughing and expelling secretions. If the lower ribs are left intact there is no danger of pressing secretions from the upper lobes down, because if they go down the patient can cough them up." Actually, however, Welles has abandoned this technic and he resects the lower ribs first (personal communication).

Alexander made (1925) an extensive survey of world statistics in order to solve this problem. He concluded that he had not found evidence that supported Sauerbruch's fear of primary upper lobe compression as the cause of postoperative pneumonia; on the contrary, reports of pneumonia following primary resection of the lower ribs are relatively frequent. He especially insisted upon the statistics of Brunner's 96 cases, many of which had been operated upon by Sauerbruch, in which seven fatal pneumonias of the compressed lung had occurred after thoracoplasties which were performed from below upwards. However, although Alexander seems to consider the physiologic integrity of the lower lobe as very useful in avoiding stasis of exudate in it, he advises avulsion of the phrenic nerve as a good preliminary measure. This appears somewhat contradictory, because the diaphragm, while an inspiratory muscle, plays an important part in cough by immobilizing the chest during the expulsive phase of cough and, especially, by allowing the air entrapped in the chest during the inspiratory phase of cough to acquire the necessary positive pressure of 60 to 80 mm. Hg (compressive phase) which renders possible the expulsion of the content of the bronchi.

I have dwelt upon the different opinions expressed on this subject rather extensively because it seems to me that so far we have been entangled in details and have overlooked the fundamentals; the fact that the statistics are essentially the same with any one of these methods is in favor

of my contention. In fact, while all agree that the fundamental factor in these compications is bronchial stasis caused by the entrapped bronchial secretions, by the exudate expressed during collapse of the diseased lung and by the pneumococcic infection of this exudate caused by organisms aspirated from the upper respiratory ways, nothing seems to have been attempted to eliminate these factors.

In closing this chapter of the study of the mechanism of production of hemorespiratory complications I shall summarize as follows the conclusions at which I have arrived and upon which the discussion of the treatment will be based.

I. The limitation of the respiratory field, due to the disease itself, to the posture of the patient during thoracoplasties, to the difficulty for adequate supervision of anesthesia and to the large amounts of bronchial exudate present, which easily obstructs the glottis, favor insufficient pulmonary ventilation and predispose to anoxemia.

II. Anoxemia is further favored by the creation of unaerated channels in the collapsed lung and in the insufficiently ventilated (because of posture and bronchial stasis) healthy lung. Rapid shallow breathing follows, washing out carbon dioxide and thus, because of the Bohr effect, further increasing anoxemia.

III. Anoxemia and acapnia cause loss of muscular tonus leading to peripheral vascular failure, insufficient thoracic expansion and lowered vital capacity; these conditions favor increased oxygen want and at the same time facilitate bronchial stasis by depriving the lung of its natural means of defense.

IV. Superinfection of the bronchial exudate by pneumococcus aspirated during the operation into the bronchi with the secretions of the throat and mouth, increases the viscosity and virulence of the entrapped exudate. Thus bronchiolar or bronchial obstructions take place, causing lobular or lobar atelectasis or pneumonia. Furthermore, by the same mechanism tuberculous infection of the parenchyma of the good lung can occur, producing spreads, tuberculous bronchopneumonias or even tuberculos pneumonias either in the healthy or in the collapsed lung.

IV. TREATMENT

(A) *Preventive.* Endotracheal anesthesia and bronchial suction.

This being the case, it is obvious that a rational preventive treatment of these complications should satisfy the following requirements:

I. During operation the patency of the respiratory ways should be insured.

II. During the whole operation sufficient amount of O_2-CO_2 should be present in the gas mixtures in order to maintain adequate oxygenation and also to prevent acapnia.

III. Any communication between the upper and the lower respiratory ways should be eliminated in order to prevent aspiration of the infected material from the former into the latter.

IV. The exudate accumulated in the bronchi or expressed out of the collapsed lung should be aspirated during the operation. The damage occurs neither before nor after but during the operation; therefore, it seems reasonable to try to remove this bronchial exudate while it is being poured out of the diseased lung into the bronchial tree.

After much thought and experimental investigation on the above requirements I came to the conclusion that endotracheal anesthesia alone could bring about a satisfactory solution of these problems. It seemed to be the only method which could insure at the same time a perfect patency of the respiratory tract during operation, a complete isolation of the lower from the upper respiratory ways (by packing the throat and mouth around the catheter) and an easy access to the bronchial tree for adequate removal of the bronchial exudate by suction.

However, the practical application of this method seemed, a priori, to present a number of serious objections. First objection was the inconvenience of the introduction of the catheter, necessitating special training and bringing in a new complication with the operation. Especially in a large sanatorium for tuberculous patients, where the patients know one another and impressions are communicated with surprising speed, it was feared that this new complicating factor would render the consents for thoracoplasty, which were already difficult, well-nigh impossible. The way to overcome this difficulty would have been to introduce the catheter after general anesthesia had been induced and this is what I did in my first cases. Rapidly it became apparent, however, that in this way we were defeating our own purposes; the laryngeal reflex is a hard dying reflex, requiring a deep narcosis for its disappearance; so that a great deal of aspiration of oropharyngeal secretions into the respiratory ways occurred during the strenuous respiratory period accompanying induction; this had been verified by introducing 10 per cent solution of india ink into the mouth of a patient and bronchoscoping him after induction was complete. Therefore we tried to pass the catheter, under local cocaine anesthesia, as a bronchoscope and we were agreeably surprised to see the relative ease of this procedure and the lack of protest from the patients. Since this method has been adopted (over a year) no patient has formulated any objection against it.

The second objection was the possibility of laryngeal irritation, by the protracted contact of the endotracheal catheter with the larynx, which could facilitate the development of tuberculous laryngitis. This problem required serious consideration as many of our cases already showed, before operation, a more or less advanced degree of tuberculous laryngitis. An inquiry was made among nose and throat specialists and the opinion of Chevalier Jackson was asked. Jackson's answer convinced me in favor of this method. In a letter addressed to Dr. Flagg, consultant anesthetist of Seaview Hospital, he stated that tuberculous laryngitis being due to infection by the lymphatics, he did not consider that local irritation would be

of any harm, and that under similar conditions he would not hesitate to advise it.

The third difficulty, purely technical, was the lack of an adequate instrument for that purpose. Magill's elastic gum, or silk or silver woven catheters cannot be easily introduced through the glottis because of lack of sufficient rigidity and they cannot be boiled. Phillips' and Flagg's intratracheal catheters are perfectly flexible, they can be transformed into rigid tubes during introduction and present a thin wall and a large diameter; but they are very easily put out of order. Because of that they become very expensive. Their passage through the glottis of nonanesthetized patients is often difficult because of the absence of illumination when the extremity of the tube has been pushed beyond the tip of the lighted laryngoscope. These difficulties have been overcome by the construction, by Dr. McKesson and myself, of a new tube the description of which is given below.

Suction of the bronchial content is an easy matter after the introduction of the endotracheal catheter. It can be performed by means of an ordinary elastic ureteral catheter or better with the specially constructed elastic catheter of Flagg, connected with any kind of suction apparatus. When an ordinary catheter is used it is advisable to cut the tip of it in order to have a terminal opening besides the lateral ones; in this way obstruction of the opening by sucking in the bronchial mucosa or by very thick exudate is avoided. Between the catheter and the tubing of the suction apparatus a sterile glass cannula or a specimen collecting tube of Tucker can be interposed, allowing visualization of the presence or absence of exudate, of its amount, its viscosity and color and rendering possible its bacteriologic examination. Upon these practical and theoretical considerations a technic has been gradually elaborated which is now giving us full satisfaction.

In the following I shall describe the instruments, the technic of introduction of the catheter, packing of the mouth and the bronchial suction, and the method of anesthesia we have developed.

Instruments:

The necessary instruments are a laryngoscope and an intratracheal catheter.

Any type of laryngoscope for bronchoscopy can be used provided its diameter is sufficient to allow the easy passage of the intratracheal catheter. Personally, I use the Flagg laryngoscope, in which the batteries are carried in the handle of the instrument.

The intratracheal catheter I use is my special endotracheal catheter* mentioned above. This catheter is composed of two solid segments, (*D* and *E*) (Fig. 4) 9 mm. in diameter, and respectively 7 and 9 cm. in length. The tubes are screwed on to either end of an intermediate and interchangeable piece (*E*) made of flexible wire coil; this coil has the same diameter as the solid segments, and measures 10.5 cm. in length. A Penrose tube is

*This catheter is constructed by the McKesson Appliance Co., Toledo, Ohio.

placed on this coil in order to make it air tight. The total length of the instrument when mounted is 26 cm.; it assumes the form of a bronchoscopic tube flexible in its central part, the distal tip of which is smoothed and slanted as in the bronchoscope of Jackson. A solid tube (*B*), of slightly smaller diameter than the catheter is introduced into the latter transforming it into a rigid bronchoscope; this internal tube is 2 cm. longer than the catheter; so that when the instrument is mounted the internal tube protrudes out of the tip of the catheter. This renders the introduction of the instrument into the glottis easier because of the small diameter of the internal tube. A light carrier is fixed inside the internal tube and the whole

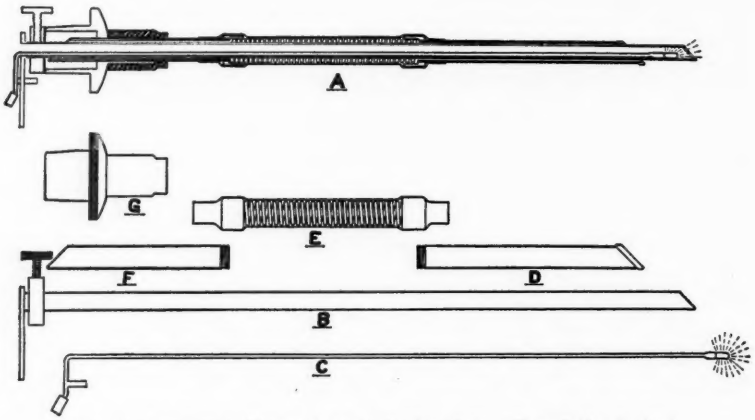

Fig. 4.—Coryllos-McKesson intratracheal catheter. Description in text.

system is immobilized by means of a screw thus rendering easy the manipulation of the whole instrument (*A*). After introduction into the trachea and packing of the mouth, a mouth gag (*G*) is slipped over the extremity of the instrument and fixed around the neck with tape; in this way the catheter is completely secured and cannot be displaced in any direction.

Technic of Introduction:

The throat is anesthetized with 10 per cent cocaine solution by the same technic as for bronchoscopy. The head of the patient is placed in the "high-low" position and the laryngoscope introduced into the mouth. The epiglottis is visualized and the tip of the laryngoscope is passed under it, allowing exposure of the glottis. When the anesthesia is complete and the head of the patient placed in the correct position, it is easy to introduce the tip of the catheter into the glottis; this is rendered easier by the addition of the light carrier to the instrument. Then the internal rigid tube is withdrawn and the laryngoscope removed while the outside end of the catheter is pushed deeper into the trachea. At that time the contact of the instrument with the trachea generally causes a coughing spell, which is rapidly relieved by injecting 1 c.c. of 10 per cent cocaine solution into the trachea

through the catheter. In all, the introduction of this catheter is easier than of a bronchoscope because once the internal rigid tube is removed, the catheter ceases to be a rigid instrument and slides into the trachea as an elastic catheter.

The criteria of successful introduction of the catheter into the trachea are two: first a full expiratory blow through the tube which is easily felt by the hand when placed near the opening of it. The second is that when the tube is in the trachea the patient cannot speak. If the patient can only say *A* with an audible voice the tube is not in the trachea; it is in the esophagus and should be removed and introduced correctly.

Packing: A gauze packing, 1½ inches in width and 3 feet long is necessary for a good packing. The beginning of the gauze packing is introduced deeply into the throat behind the catheter. The whole oral cavity is methodically packed. The mouth gag is then slipped over the oral end of the tube and secured in place by tapes lightly tied around the neck.

Suction: The elastic catheter connected with the suction apparatus is then introduced into the intratracheal catheter and the bronchi are aspirated before anesthesia is started; effort is made to remove all exudate that can be removed; there is no danger whatever of pushing this catheter deeply into the bronchial tree.

During the operation the suction is repeated in the same way, temporarily disconnecting the intratracheal catheter from the anesthesia apparatus. The frequency of the suction depends on the amount of secretion present in the bronchi, usually every 5 to 15 minutes. Five to thirty cubic centimeters of viscid secretion are removed during the operation.

At the end of the operation the patient is placed in the dorsal decubitus and the bronchi are aspirated again; the endotracheal catheter and packing are then removed and the throat is carefully sucked out with an ordinary suction metallic aspirator.

Anesthesia: The endotracheal catheter can be connected to any kind of anesthesia apparatus, provided that a rubber bag is interposed between the two, in order to equalize pressure and to allow a good mixture of gases and rebreathing. It can also be used for inhalation anesthesia by directly connecting the tube to an ether can by means of a long rubber tube, as in the method described by Flagg, or even when local anesthesia is used.

Personally I prefer the recording anesthesia apparatus of McKesson, because of a number of its special features which appeal to me. The recording of the tidal air allows me to look at the lever from time to time during the operation and to know if my patient breathes and how he breathes. The possibility of having an accurate record (Figs. 5 and 6) of the mixtures of gases used, of the variations of blood pressure and of the different operative acts, incidents or accidents, helps and is particularly instructive on going over these charts later on. McKesson presented a good summary of the information given by these records at the last annual convention of the American Anesthetists' Association (1932).

As anesthetic the "mixture" gas-oxygen-ether is being used now. The amount of ether generally used during the forty minutes, which is the average duration of our thoracoplasties, varied in our cases between one-half and one ounce. The anesthetic mixture is administered under slightly positive pressure +4 to +6 mm. Hg. The objective of positive pressure is threefold; it keeps the lungs in a constant degree of moderate inflation which prevents sudden collapse in case of accidental opening of the pleural cavity; it increases the partial pressure of oxygen in the alveolar

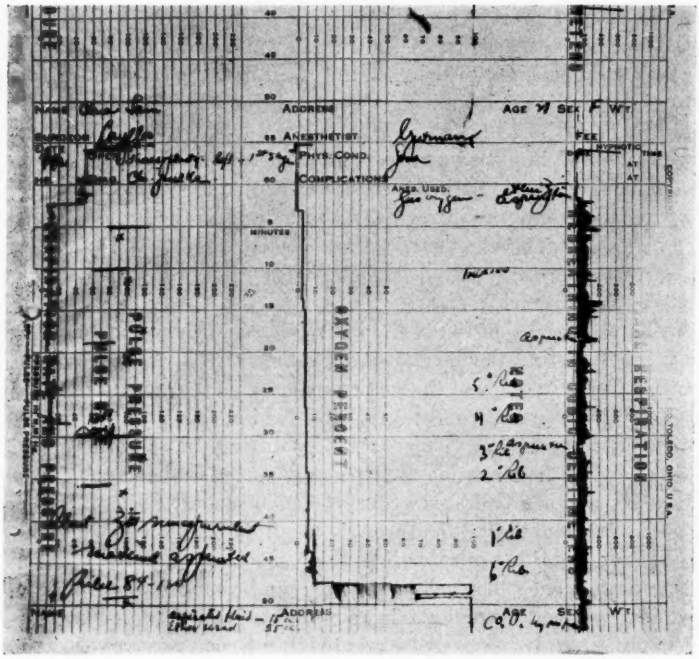

Fig. 5.—Specimen of anesthesia chart. (Recording anesthesia apparatus of McKesson) first stage thoracoplasty from the first to sixth rib.

The short horizontal lines in the left represent the blood pressure, and the crosses the pulse rate. The vertical line in the middle indicates the percentages of the nitrous-oxide-oxygen mixture. In the left is the tracing of the respiratory movements. The interruptions in the latter correspond to bronchial suctions.

The total duration of operation, the time of the resection of the different ribs, of bronchial suctions and of any incident or accident occurring during the operation are shown on this chart.

air and creates a constant draft from inside out, which, with the packing of the throat prevents any aspiration of oropharyngeal secretions.

At the end of the operation, when the suturing of the wound is started, a mixture of 10 per cent CO_2-O_2 is substituted for the anesthetic mixture, causing marked hyperventilation. This procedure, as Henderson and Haggard, Birnbaum and I have insisted upon, acts by increasing the volume of the air brought into the lung thus assuring a better ventilation

of a greater portion of the lung parenchyma. A. Lincoln Brown has given valuable information on the action of CO_2 by observing the behavior of the bronchi through a bronchoscope in a case of atelectasis while CO_2 was being administered. He noted: distinct blanching of the mucous membrane of the trachea and of the bronchi, the production of violent movements of the bronchial tree and alteration of the shape of the small bronchi thereby tending to free adherent mucus. More recent and yet unpublished experimental work of Birnbaum and myself, seems to indicate that CO_2, by dis-

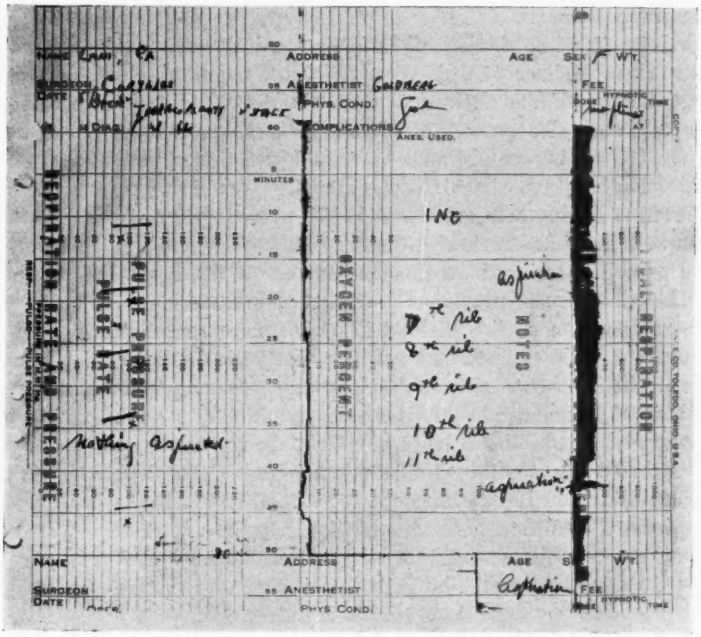

Fig. 6.—Same patient as in Fig. 5. Second stage thoracoplasty, from seventh to eleventh rib.

solving in the exudate as H_2CO_3, shifts the P_H of this exudate to the acid side, often as low as 6.0. It is thus possible that when O_2-CO_2 inhalation is prolonged the development of the pneumococcus is restrained or even inhibited by the acidity of the medium. At any rate, it hastens the elimination of the anesthetic gases dissolved in the blood so that the patient is out of anesthesia at the end of the operation. Anesthetists with special experience in thoracic surgery, such as H. R. Griffith and more especially Hewer and Grudner, have insisted upon the importance of the awakening of patients from anesthesia as soon as possible after operation, in order to early regain their cough reflex. "The shorter the postoperative sleep the lesser the danger" says Grudner. It is a routine test on my services that

the patient must be able when he leaves the operating room to be sufficiently awakened to tell *his name and the date of operation.*

A Few Remarks on Endothoracic Anesthesia:

Endotracheal administration of anesthesia combined with suction is a valuable adjunct to our surgical technic in chest diseases. It gives us perhaps the missing link between preoperative and postoperative care of these patients; for, if I am correct in my theoretical considerations, I believe that complications occurring during the early postoperative period have been initiated during operation, and that they are closely connected with bronchial stasis.

Endotracheal anesthesia is not a new procedure. After the epoch making work of Meltzer (1910), Auer, Elsberg, Leighton, Peck, Giordano, Pratt, Saltzer, Evans, Robinson, Cotton and Bothby and more recently Adson, Jackson, Hargrave, Rowbotham, Moss and more especially Waters, Hewer, Hepp, Magill and Flagg, have submitted this method to careful investigation both experimental and clinical. Over 10,000 cases have been reported as having been submitted to this procedure. A great variety of apparatus and intratracheal catheters have been invented. This method has emerged victorious from this long and severe test in which it has been used both as an insufflation or as an inhalation anesthetic procedure.

A modification of this method has been more recently presented by Waters. It is the "closed endotracheal anesthesia" (1932), in which the intratracheal catheter is introduced into one common bronchus and secured in an air-tight manner to the trachea by inflation of a small annular balloon applied around the catheter, similar to the one used by Birnbaum and myself in our investigation upon gas exchanges in the obstructed bronchus. The oral end of the catheter is connected with a closed anesthesia apparatus containing a soda-lime cannister which absorbs CO_2; in this apparatus the initial volume of nitrous-oxide or ethylene circulates from the lungs to the apparatus and back to the lungs and only oxygen in small amounts equal to the metabolic needs of the organism is added.

Preoperative and Postoperative Care:

(a) *Preoperative Care:*

As we do not as yet possess any dependable "functional test of the cardiorespiratory system" similar to the functional renal test, we should proceed with caution in the selection of our cases. Hemoglobin below 70 per cent, considerable increase of respiration and pulse rate after slight exercise, low blood pressure, toxic symptoms and too great mobility of the mediastinum are the important signs of unfitness of the patient for thoracoplasty. Hemoptysis, moderate gastrointestinal or kidney involvement, and even bilaterality of lesions, provided that the lesion is at least on one side limited to the apex, are not absolute contraindications provided that the requirements above mentioned are satisfied.

Digitalization of the heart of the patient before operation does not seem to be of any great value. Moderate exercise during the days preceding

operation is advisable although our patients for thoracoplasty, as a rule, are patients to whom some degree of activity was allowed.

Of greater importance is to instruct patients to completely empty their bronchi before operation, and special attention to this point should be given during the hours preceding operation. No narcotics impairing the action of the respiratory center or cough are given before operation such as morphine, atropine or scopolamine. We have found that amytal or luminal by mouth four hours before operation in a dose of four grains does not exert any untoward effect upon respiration. Ephedrine (one ampoule) is as a rule given before operation and strychnine (1/60 to 1/30 of a grain or more) and caffeine-sodium-benzoate are administered during operation if necessary.

Two other methods are routinely taken during operation. Diathermic heating of the patient during the operation and glucose-saline infusion, the second only when a long operation upon a weak patient is contemplated.

(B) *Curative Treatment and Postoperative Care:*

With the use of intratracheal anesthesia and bronchial suction the postoperative course became much smoother; shock and especially respiratory embarrassment and high temperatures have decreased in a very considerable degree. The postoperative curves of temperature; respiration and pulse rates, and of blood pressure showed very marked improvement. These differences are especially marked in the "wet" cases, i. e., patients expectorating over 15 c.c. in 24 hours, and less in the "dry" cases. Unfortunately it is not always possible to predict with any degree of certainty from the amount of sputum expectorated in 24 hours the amount of bronchial material which can be expelled from the collapsed lung during operation. As a rule, however, we can state that dry cases can be operated upon with any kind of anesthesia whereas in wet cases special care should be taken for a proper bronchial drainage. Dangers of hemorespiratory complications had been by this method greatly lowered, but they have not been as yet completely eliminated. In fact complete bronchial aspiration is not always feasible, nor can bronchial stasis by exudate accumulated in the bronchi subsequent to the operation be safely avoided; disturbances due to anoxemia can develop at any time during the 24 or 48 hours following operation. Therefore a constant and vigilant postoperative supervision and care are necessary and more so than in any other surgical operations. Time and again patients looking perfectly well after operation have presented within a short time the most severe cardiopulmonary complications, and, conversely, patients apparently and practically dead were restored to life and to health. We can summarize as follows the postoperative measures adopted in our postoperative cases.

The patient is kept 4 to 12 hours in shock position, lying on the operated side. In all cases CO_2-O_2 (7 to 10 per cent) is given continuously by means of a nasal catheter for 2 to 4 hours after operation and subsequently as indicated by the condition of the patient. It must be constantly borne in

mind that it is easier to prevent than to cure postoperative pulmonary complications and it should never be forgotten that even a pulmonary complication which does not kill the patient exposes him to postoperative tuberculous spread.

The patient should be encouraged to cough and expectorate by his nurse who helps him by supporting his chest. I no longer use any compression corsets or other immobilizing devices on the patients; on the contrary the patient should be turned from side to side, as soon as his condition allows it, with all necessary precautions to avoid any excessive disturbance and pain. Often patients cough and expectorate only when placed on the healthy side, as in postoperative atelectasis. In fact, immobilization of the patient on the diseased side does not avoid passage of secretions to the healthy lung. This takes place when the bronchi of the diseased lung are filled up with mucous exudate which little by little flows into the healthy bronchi.

Narcotics as morphine and codeine are used in small doses, and only if absolutely necessary, so as to reduce pain without inhibiting cough and impairing the respiratory center.

Pulmonary complications, patchy or lobar atelectasis which may or may not go on to clinical bronchopneumonia or pneumonia and which are always related to bronchial occlusions, generally develop within 24 hours following operation. As a general rule, the first measure to combat these complications is transfusion of 500 c.c. of whole blood. It is unwise to procrastinate and postpone this measure under the pretext that the condition of the patient does not appear to be sufficiently bad. It is the only rational method to instantly relieve anoxemia and this is the purpose for which it is given. Fall of blood pressure, rapid pulse rate, shallow breathing, cold sweats, cold extremities with or without cyanosis, in a word the syndrome attributed to "heart failure," is but the result of acapnia and anoxemia. The involvement of the lung does not allow emergency relief of CO_2-O_2 want through this organ. A transfusion of 500 to 700 c.c. of blood, on the contrary, will bring rapidly and directly into the circulating blood the necessary amounts of CO_2-O_2. Loss of muscular tonus, peripheral vascular failure and anoxemia of the respiratory, circulatory and vasomotor nervous centers will be instantly relieved. This is, I believe, the reason for the instantaneous and often miraculous changes occurring in these patients, which cannot be explained by a mere mechanical elevation of blood pressure. Peripheral vascular failure produces the same disturbances as hemorrhage; the patient "bleeds into his own vessels" so that the amount of blood returning to the right heart is greatly decreased. It is even worse than ordinary hemorrhage for to the decrease in the amount of circulating blood and of its oxygen carrying capacity is added the increased need of oxygen in the tissues because of blood stasis and the deficient oxygenation in the involved lung. In fact, as pointed out above, a careful observation of the patient in an anoxemic crisis to whom whole

blood is administered shows successively: decrease of cyanosis when present, slower and deeper respiratory movements and after several seconds only a slower and fuller pulse. Another phenomenon repeatedly noted is that patients in very desperate condition and unable to cough, have started coughing during or shortly after transfusion; they have expectorated viscid sputa which placed in water proved to be bronchial casts and this was followed by a really spectacular improvement in their condition, exactly as in atelectasis. The explanation we offer of this fact is that relief of CO_2-O_2 want has restored muscular tone, allowing stronger action of inspiratory and expiratory movements, better action of the diaphragm, greater inspiratory dilation of the bronchi and more efficient bronchial peristaltic movements; the immediate results were increased vital capacity, more air-storage in the lung, displacement of mucus obstructing the bronchi, and creation of airways between the occluding mucus and the bronchial walls allowing air to penetrate into the alveoli and causing efficient expulsive action of cough.

Cardiotonics, such as digifoline, caffeine or lobelia extract have been of little help. Strychnine on the contrary, grains 1/60 repeated every four or even two hours and ephedrine or adrenalin in repeated small doses of 5 to 10 minims and coramine have been very helpful.

In closing I want to insist again upon two points. First, that these patients should never be left alone during the first 48 hours after operation. Anoxemic death can occur within a few minutes after very little or even no warning on the part of the patient, and only prompt action by a specially trained personnel can prevent it. Second, that we should never lose hope as long as the heart is beating. A striking example of what can be done in similar cases is given by the following case.

W. S., white female, thirty-two years of age. Metropolitan Hospital of the City of New York. Caseous pneumonic tuberculosis of the left lung.

Because of damage of the intratracheal tube ordinary gas-oxygen-ether (mask) anesthesia was administered. An upper stage thoracoplasty was performed. Five ribs were resected, from the first to the fifth. Moderate shock at the end of the operation. Seven hours after operation patient suddenly became dyspneic and within ten minutes condition grew desperate. Extremities became cold, body was covered with cold sweat, pulse was not any longer perceptible, respiration stopped, and corneal reflexes disappeared. Heart beats hardly heard.

Artificial respiration, insufflation of O_2-CO_2 and intracardiac adrenalin injection was given, and a transfusion was started immediately. The condition only slightly improved, when, at the end of the transfusion patient started coughing and expectorated about 5 c.c. of very viscid sputum, which when placed in water proved to be a large bronchial cast. She showed at once a spectacular improvement and within a few minutes her condition was completely transformed. She made an uneventful recovery. Four weeks later the same patient had a second stage thoracoplasty (6 ribs) without the slightest postoperative complication. A spread in the healthy lung, exudative in nature, produced after the first stage resolved entirely. She was discharged from Metropolitan Hospital four months after the second stage and she remains since that time (March, 1932), in perfect health, cavities closed and sputum persistently negative.

Similar cases are neither exceptional nor rare. Had we tried to resuscitate this patient by the usual cardiotonic injections we should have certainly failed and death would have been assigned to "heart failure," "acute cardiac dilation" or to embolism. In reality it would have been caused by acute anoxemia due to sudden obstruction of a major bronchus. Blood transfusion, by relieving the anoxemia, even temporarily, restored muscular tonus and rendered possible cough, which brought about the expulsion of the bronchial plug and caused the immediate suppression of the cause of anoxemia. Increasing experience in thoracic surgery, especially in pulmonary tuberculosis, teaches the importance that competent and vigilant postoperative care have in the final results.

IV. STATISTICS AND RESULTS

As a basis for the discussion of the results obtained by intratracheal anesthesia, suction method, I shall take all cases of thoracoplasty for pulmonary tuberculosis operated upon in our divisions of thoracic surgery in Metropolitan and Seaview Hospitals with intratracheal anesthesia (Table III).

From September, 1931, until October, 1932, 133 tuberculous patients were submitted to thoracoplastic collapse representing 250 operations. *All these cases, without any exception presented cavities, and belonged to the ulcero-caseating type, or better to the caseous pneumonic form of Ornstein, Ulmar and Dittler.* In a number of them productive changes were present in the contralateral lung, and in 3 cavities were present in the other apex. As it has been explained elsewhere, the former can and should be considered as unilateral cases.

Of these patients, 84 were operated upon with intratracheal anesthesia, while 49 were operated upon with ordinary mask anesthesia. In both groups the same gas-oxygen-ether mixtures were used. The surgical work in the two hospitals composing our tuberculous center is carried on by two surgical staffs, shifting from one to the other hospital every four months. In that way the operative material of both staffs is identical as it is drawn from the same hospitals and selected in surgical conferences by the same medical and surgical staffs. On the other hand the operative technic used by both staffs is approximately the same. In all patients operated upon by my staff intratracheal anesthesia was used, whereas in the patients operated upon by the other staff ordinary mask anesthesia was used. It is obvious, therefore, that the patients of the second group can serve as control for the first group.

Table III gives a résumé of the results obtained in each of these two groups.

The differences in the morbidity in the two groups are really impressive (9.5 per cent with intratracheal against 20.4 per cent with ordinary anesthesia). The mortality, although lower with intratracheal-suction anesthesia (15.5 per cent in group A against 18.3 per cent in group B) does

TABLE III

	GROUP I GAS-OXYGEN-ETHER INTRATRACHEAL INSUFFLATION-SUCTION						GROUP II GAS-OXYGEN-ETHER MASK ANESTHESIA					
	METROPOLITAN HOSPITAL 33		SEAVIEW HOSPITAL 51		TOTAL 84		METROPOLITAN HOSPITAL 6		SEAVIEW HOSPITAL 43		TOTAL 49	
	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT
I												
General Results:												
Cured (persistently negative sputum)	16	48.4	30	58.8	46	54.8	1	16.6	11	25.5	12	24.5
Greatly Improved (Gaffke 0-2)	10	30.2	3	5.8	13	15.5	1	16.6	7	16.3	8	16.5
Not Improved (Gaffke above 2)	0	0	7	13.8	7	8.3	1	16.6	18	41.8	19	38.7
Deaths:												
I. Within 3 weeks, all causes	5	15.1	8	15.7	13	15.5	3	50.0	6	13.9	9	18.3
II. After 3 weeks, all causes	2	6.2	3	5.8	5	5.9	0	0	1	2.4	1	2.0
Deaths due to the operation itself	1	3.3	5	9.8	5	7.1	3	50.0	5	13.9	8	16.5
II												
Morbidity:												
Postoperative cardiorespiratory complications	2	6.4	6	11.7	8	9.5	3	50.0	9	21.0	12	20.4
III												
Patients discharged in 3 to 7 months	10	33.3	17	31.3	27	32.1	1	16.6	4	9.3	5	10.2

not keep the same pace with morbidity. This is apparently due to the fact that a number of extraneous causes had caused death in group I of the Metropolitan Hospital which have to some extent altered the results. The following are the causes of death on the charts of the patients.

1. Shock.

1. Cerebral hemorrhage, 5th day, after uneventful postoperative course, in a colored woman with positive Wassermann.

1. Miliary tuberculosis (autopsy).

1. Wound infection. Extensive amyloidosis (autopsy).

1. Thyrotoxicosis. Sudden death, 9th postoperative day in a woman, 40, with marked hyperthyroidism.

On the other hand, the three deaths of the Metropolitan Hospital Group II operated on with ordinary anesthesia, were due to:

1. Atelectasis

1. Pneumonia

1. Bronchopneumonia

A similar analysis of the Seaview Hospital group, shows that in the intratracheal anesthesia group, death was due:

2. Postoperative pneumonias

1. Acute cardiac dilatation

1. Shock

1. Displacement of the trachea to such an extent that bronchoscopic examination showed kinking and almost complete closure of the main bronchus to the operated side.

2. Infection of wound.

1. Three months after operation streptococcic infection of arm.

1. Diabetes with hypertension, two months after operation. She had completely recovered from operation, sputum was negative and she had been discharged from the surgical division.

1. Chronic obstruction of the common duct possibly cancer of the pancreas or liver. She died three months after operation. Sputum had been persistently positive. Autopsy refused.

1. Bilateral case of pulmonary tuberculosis.

Analysis of the group of patients operated upon in the same hospital with ordinary mask anesthesia shows, that excepting two (one died with tuberculous meningitis and another with prolonged pleural suppuration) the others died from cardiorespiratory complications, one with postoperative pneumonia, two with shock and one with cardiac deficiency.

Moreover, we find in the same group three cases which developed postoperative atelectasis, all in the lower lobe of the operated side. These cases recovered with prolonged oxygen-carbon dioxide inhalations and transfusions of whole blood.

Another point of importance, upon which I have already insisted, is the course of the disease itself after thoracoplasty. In the group of 84 patients operated upon with intratracheal insufflation-suction anesthesia we find

that 54.8 per cent became bacilli free and 15.5 per cent almost bacilli free; in the latter, in repeated examinations only occasionally very few scanty bacilli could be discovered (Gaffke 0-2). In other words of all the patients with cavities operated upon 70.3 per cent were restored to health.

The difference is striking in the second group of 49 patients operated upon with ordinary mask anesthesia. Of these patients only 24.5 per cent became bacilli free and 16.5 per cent had occasional positive sputum with very scanty bacilli (Gaffke 0-2). That is 51.0 per cent, as compared with 70.3 per cent of the previous group, were restored to health. As against these figures we have the numbers of nonimproved patients. In the intratracheal anesthesia group only 8.3 per cent remained stationary as compared to 38.7 per cent of the second group.

The comparison of the number of patients discharged after thoracoplasty in the two groups is equally impressive. In the first group 32.1 per cent of all patients operated upon had been discharged within three to seven months after operation, whereas in the second group 10 per cent were discharged.

It is obvious that the technic used by the surgeons operating in each group being approximately the same, the patient being drawn from the same hospitals and selected by the same group of medical men at the same surgical conferences, the above striking differences in the results obtained cannot be considered as mere coincidences. On the other hand, the number of patients presented in this statistical study is large enough to exclude the possibility of a streak of good or bad luck. These 133 cases represent 250 operations; the number of operations is even more important than the number of patients because complications and deaths do occur in every operation and with the same frequency.

These results have so far corroborated the soundness of the working hypothesis which has been behind this investigation. Although it is too soon to elaborate definite statements, the results appear sufficiently substantial as to be presented to our Society. Personally, I shall keep on, checking up on the course of the operated cases and reporting them to you as time goes on. The problem is of importance and the reward in the saving of human lives is worthy of the effort.

SUMMARY AND CONCLUSIONS

Clinical and experimental investigation on the etiology, prevention and treatment of pulmonary and cardiac complications following thoracoplasties for pulmonary tuberculosis was carried on in two Institutions of the City of New York, composed of 2,000 beds for tuberculous patients.

The results reported in this paper were based on 133 patients upon whom 250 thoracoplastic operations were performed from August, 1931, to October, 1932.

Clinical and experimental evidence was presented showing that these cardiopulmonary or more exactly hemorespiratory complications are due

to respiratory and circulatory deficiency, causing acute or prolonged O_2-CO_2 want and leading to anoxemic crisis and death, if not prevented in time.

The initial cause of these deficiencies is stasis of the bronchial exudate present in the lung before operation and especially of exudate expressed during operation from the collapsed, diseased lung. Infection of this exudate by pneumococcus, always present in the upper respiratory ways, increases its viscosity and renders it able to obstruct small or large size bronchi. Lobular, lobar or massive atelectasis, bronchopneumonias or pneumonias may develop which further increase anoxemia by decreasing the respiratory field, by creating unaerated channels in the lung, and by increasing the dead space in this organ; the resulting shallow rapid breathing further increases anoxemia, causes massive elimination of CO_2 and produces acapnia. The latter still increases anoxemia, and, furthermore, it brings about loss of muscular tonus; the latter causes "peripheral vascular failure," peripheral blood stasis, decrease of the venous return to the heart, drop of blood pressure and "shock," and leads to more marked anoxemia and to anoxemic crisis.

Deaths attributed to aspiration or tuberculous pneumonias, or to heart failure, cardiac dilatation or shock have really been at last analysis anoxemic deaths.

Preventions of these complications could only be possible if this vicious circle could be stopped at its origin. Satisfied that this vicious circle begins during the operation we elaborated a method of administration of anesthesia which fulfills the following requirements: it keeps the respiratory ways patent, suppresses communication between the upper and the lower respiratory ways, allows an adequate ventilation of the lungs and, above all, it renders possible repeated suction of the bronchi and elimination of their content, and this before, during and after operation. A method of intratracheal insufflation anesthesia and bronchial suction was thus evolved. It has been used in 84 cases, representing 152 operations. In the other 49 cases (98 operations), ordinary mask anesthesia was used. All other conditions in these two groups having been equal the latter group served as control for the first, allowing a comparative study of this method. Although definite statements cannot yet be formulated, there is strong evidence in favor of the intratracheal anesthesia method combined with bronchial suction.

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Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts

Mode of Action of Respiratory Stimulants: I. Mode of Action of Oxygen Lack. S. Selladurai and Samson Wright. Quart. J. Exper. Physiology **22**: 233, 1932.

The authors review the several theories on the effect of oxygen lack on respiration. They mention Rosenthal's theory of increase in the responsiveness of the respiratory center to carbon dioxide and the theory of Herman that oxygen lack causes an accumulation of circulating incompletely oxidized metabolites which act upon the respiratory center. These and other theories have one feature in common—that they assume that anoxemia acts by modifying the chemical environment of the center itself.

A new point of view emerged from the work of Heymans and Heymans in 1927. They showed that chemical changes in the blood of the trunk acted through the vagi to stimulate the respiratory center in the otherwise isolated brain.

The authors in reexamining the subject carried out experiments (1) on decerebrate animals and (2) on intact animals under chloralose anesthesia. Quantitative determinations of pulmonary ventilation were made by allowing the animal to breathe through Müller valves, the expired air being collected in a series of small bags. Qualitative records of respiration were obtained by means of a balloon inserted between the upper surface of the liver and the diaphragm and connected with a Marey tambour. The effects of inhalation of concentrations of oxygen ranging from 4 to 16 per cent and of nitrogen were studied.

With inhalation of 16 per cent of oxygen mixture, in the decerebrate cat with carotid sinus nerves and vagi intact, or with sinuses denervated and vagi intact, inhalation of this mixture produced little change in breathing. When the vagi were divided also, the inhalation inconstantly produced a slight decrease in the pulmonary ventilation.

Oxygen mixture, 11.5 per cent, brought about respiratory failure in a comparatively short time.

Oxygen mixture, 7.5 per cent, regularly stimulated respiration when the afferent nerves were intact. In five animals the respiration increased from 11 to 85 per cent. With the vagi divided, this grade of

anoxemia produced in some instances a sustained increase in pulmonary ventilation.

Four per cent oxygen mixture produced practically the same effects as the 7.5 per cent mixture.

The inhalation of nitrogen with the nerves intact usually led to an initial increase in the depth of breathing; this was followed by the sudden onset of panting. The breathing then failed. After elimination of the buffer nerves there was usually a transient increase in ventilation before failure occurred.

Under chloralose anesthesia concentration of 9.6 and 7.6 per cent oxygen was tolerated for ten minutes or longer. The stimulating action of such concentration was not as marked as in the decerebrate preparations. With the carotid sinuses denervated and the vagi divided these grades of anoxemia again caused rapid failure of breathing. With the vagi divided and the carotid sinuses intact these degrees of anoxemia were withstood in the chloralosed animal as long as twenty minutes. After denervation of the sinuses anoxemia produced the usual rapid respiratory failure.

The effects of anoxemia on blood pressure were studied to determine to what extent the respiratory changes were the result of alterations occurring primarily in the circulation. It was found that the blood pressure might remain unchanged until respiratory failure occurred. With the cessation of breathing the pressure usually dropped rapidly. After the initial steady period it sometimes fell before breathing ceased altogether. In the decerebrate animal the blood pressure sometimes rose for a short time prior to the onset of respiratory failure. These results indicated to the authors that there is no constant relation between the onset of respiratory failure and the actual level of the blood pressure or the degree of fall of the blood pressure. They infer that the fall of blood pressure when it did occur was not a dominant factor in the production of respiratory failure by anoxemia.

Their conclusions are as follows:

1. With the sinus nerves and vagi eliminated, oxygen lack acts as a depressant to the "isolated" respiratory center. The increase in breathing which is frequently obtained in the intact animal is wholly reflex and depends on excitatory afferent impulses in the sinus nerves and the vagi which overcome the direct depression of the center which is simultaneously occurring.

2. The reflex stimulation from the sinuses may, under certain circumstances, be of greater importance than that produced by the vagi.

3. The integrity of the carotid sinus nerves and the vagi may enable the respiratory center to withstand for long periods degrees of anoxemia which are lethal to it when it has been "isolated" by section of the vagi and denervation of the sinuses.

4. The classical theories accounting for the stimulating action of

oxygen lack are reviewed, and it is suggested that in the light of the fresh evidence they are no longer tenable. Anoxemia decreases the response of the "isolated" respiratory center to carbon dioxide.

Joseph A. Weinberg.

Mode of Action of Respiratory Stimulants: II. Mode of Action of Carbon Dioxide. S. Selladurai and Samson Wright. Quart. J. Exper. Physiology **22**: No. 3, 1932.

It has generally been assumed that the influence of carbon dioxide on breathing depends entirely upon its action on the respiratory center. But there is recent evidence that to some extent the respiratory effects of carbon dioxide may be produced reflexly. The authors cite the work of Heymans and Heymans in which they concluded that the afferent endings in the aortic arch could be affected by changes in the carbon dioxide concentrations and the reaction of the blood, and in consequence reflexly modify respiration. However, Gesell was unable to confirm these results.

The authors studied the effects of denervation of the carotid sinuses. It appeared from their work that in the cat the chemical composition of the blood in the sinuses normally exerted a tonic excitor effect on respiration.

The effect of carbon dioxide on the nerves of the carotid sinuses was studied by closing and opening the carotids with inhalation of carbon dioxide. It was concluded that the greater reaction obtained when the sinuses were intact was due to the excitor impulses set up in the sinuses by the alteration in blood resulting from the raised CO_2 concentration.

Effects of vagotomy were also studied. In the intact animal double vagotomy had a variable effect on resting ventilation. In the various animals it was increased, decreased or remained unaltered. With breathing of carbon dioxide in animals under chloralose with sinuses intact, double vagotomy had no effect on the response to CO_2 in four cases and reduced the reaction in a fifth. With sinuses denervated double vagotomy caused an increased response varying from 15 to 130 per cent. In decerebrate preparations the results were erratic as were the results of the decerebrate preparations with the intact sinuses.

They arrive at the following conclusions:

1. In the cat either decerebrate or under chloralose anesthesia denervation of carotid sinuses usually decreases the rate of breathing and the minute ventilation. The same result is obtained if the vagi are cut previously. It is concluded that the chemical composition of the blood stimulates the sinus nerve endings in the resting animal to discharge tonic excitor impulses to the respiratory center.

2. The response to CO_2 is usually reduced by denervation of the sinuses in the intact or vagotomized animal. It is concluded that the altered condition of the blood resulting from the raised CO_2 concentration stimulates respiration in part reflexly from the carotid sinuses.

3. Vagotomy produces variable effects on the resting ventilation and on the response to CO_2. In animals under chloralose with sinuses denervated vagotomy frequently causes an enhanced reaction to CO_2. It is suggested that CO_2 may act differently on the various sensory components of the vagus nerve.

4. As a rule the response of the intact animal to CO_2 is greater than that of the same animal with sinuses denervated and vagi divided.

Joseph A. Weinberg.

Evans, Arthur: A Rubber Esophagus. Brit. J. Surg. **20**: 388, 1933.

The author reports a case in which the larynx, part of the pharynx, the cervical esophagus and a part of the thoracic esophagus were removed twenty-three years previously for extensive carcinoma. The patient was alive and in good health at the time of the report and was able to take all food by mouth, masticate and swallow it without untoward results. The patient was a forty-year-old woman who gave a history of hoarseness and difficulty in swallowing solids of five months' duration. Examination revealed a large tumor involving the pharynx and larynx, but no obvious gland enlargement. A preliminary gastrostomy was performed on September 28, 1909. Through a midline incision from the hyoid to the suprasternal notch and a transverse incision at either limit the contents of the anterior compartment of the neck were exposed and the malignant mass was removed. Convalescence was quite uneventful. The patient now had permanent tracheal and pharyngeal openings. By a subsequent plastic operation on the neck, the opening of the sinus leading from the pharynx was moved upward beneath the hyoid bone. The short end of a rubber empyema tube was then passed into the upper pharynx, using the largest that could be introduced, and secured by a piece of tape passed around the neck. The long end of the empyema tube was connected to the gastrostomy tube.

At the onset, "the swallowing tube did not act well," wrote the patient. But by the end of a few months it functioned very well. The patient gained much weight and strength and was able to lead quite an active life. The original empyema tube was replaced by a curved rubber tube fitted with a flange 2½" from the tip introduced into the pharyngeal sinus. The original Jacques catheter No. 9 was replaced by a rubber tube which was increased in size from time to time until the present one in use is of red rubber tubing bore No. 15 and is fitted with a flange 5" from its lower end. It is secured by tapes tied around the abdomen.

W. E. Adams.

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The American Association for Thoracic Surgery

Washington, D. C., May 9, 10, 11, 1933

Hotel Headquarters—The Willard Hotel

Program

Tuesday, May 9

9:00 a.m. National Museum Building.

1. Business Meeting of the Association.
2. The Results of Phrenic Nerve Operations in 225 Cases With a Discussion of the Technique of the Operations.
   Dr. H. Ryerson Decker, Pittsburgh.
3. An Estimate of the Value of Phrenic Nerve Interruption for Phthisis Based on 654 Cases.
   Dr. John Alexander, and Dr. Lawrence Nehil, Ann Arbor.
4. Experimental Study of the Effect of Phrenicectomy on Cough.
   Dr. Herbert A. Carlson, Minneapolis, Dr. Harry C. Ballon, Montreal, Dr. Hugh M. Wilson and Dr. Evarts A. Graham, St. Louis.
5. Thoracoplasty.
   Dr. E. J. O'Brien, Detroit.
6. The Operative Mortality From Thoracoplasty in Pulmonary Tuberculosis. Analysis of 6 Fatal Cases.
   Dr. H. L. Beye, Iowa City.
7. A Study of 150 Cases of Thoracoplasty for Pulmonary Tuberculosis During the Past Two Years—Reflection of Technique and Results.
   Dr. Pol. N. Coryllos, New York City.

8. Bronchogenic Carcinoma. Special Reference to Its Classification, Prognosis and Treatment.
   Dr. Louis H. Clerf and Dr. B. L. Crawford, Philadelphia.

1:00 p.m.

1. Collapse Therapy and the Ambulatory Patient.
   Dr. J. A. Myers, Minneapolis.
2. The Future Surgical Status of the Collapse Therapy Patient.
   Dr. T. J. Kinsella, Oak Terrace, Minn.
3. Pneumocavernolysis in the Treatment of Pulmonary Tuberculosis With Cavitation.
   Dr. Harold Neuhof, New York City.
4. Non-Penetrating Wounds of the Heart. The Cardiac Manifestations Brought About by Acute Peripheral Compression.
   Dr. Claude S. Beck and Dr. Ernest Bright, Cleveland.
5. Removal of Needle From the Heart With Electrocardiographic Studies Before, During, and After Operation.
   Dr. Francis Scrimger, Montreal.
6. The Diagnosis and Treatment of Encapsulated Empyemata.
   Dr. W. A. Hudson, Detroit.
7. Moving picture study of the treatment of acute empyema.
   Dr. Jos. Gale, Madison.
8. Moving picture study of the treatment of acute empyema.
   Dr. Ralph B. Bettman, Chicago.

Wednesday, May 10

9:00 a.m.

1. Executive Meeting of the Association.
2. President's Address.
   Dr. George P. Muller, Philadelphia.
3. Address on Lung Abscess and Pleural Effusion.
   Dr. Marc Iselin, Paris, France. (By special invitation.)
4. Pneumonectomy for Sarcoma of Lung in a Tuberculous Patient.
   Dr. Howard Lilienthal, New York City.
5. Dermoids of the Mediastinum.
   Dr. Carl A. Hedblom, Chicago.
6. Intrathoracic Teratomas.
   Dr. S. W. Harrington, Rochester, Minn.
7. Esophagectomy for Cancer of the Upper Esophagus With Lessons Derived From an Operative Failure.
   Dr. P. E. Truesdale, Fall River, Mass.
8. Experiences With Oleothorax Treatment.
   Dr. John N. Hayes and Dr. Lawrason Brown, Saranac Lake.

1:00 p.m.

1. Active Contractility of the Bronchopulmonary Smooth Muscle as Demonstrated by Electrobronchographic Records.
   Dr. Ethan F. Butler, Elmira, N. Y.
2. An Experimental Study of the Reactions of the Pleura to the Bacillus of Tuberculosis.
   Dr. W. S. Lemon, Rochester, Minn.
3. Effects of Laparotomy and Abdominal Distention on the Lung Volume.
   Dr. Edward D. Churchill, Dr. H. Beecher, Dr. H. H. Bradshaw, and Dr. G. E. Lindskog, Boston.

4. The Treatment of Traumatic Haemothorax. An Experimental and Clinical Study.
   Dr. D. C. Elkin and Dr. J. C. Sandison, Atlanta.
5. Physiological Determinants in Surgery for Relief of Cardiac Pain.
   Dr. Peter Heinbecker, St. Louis.
6. The Tensile Strength of the Paralyzed Diaphragm.
   Dr. Richard H. Meade, Jr., Philadelphia.
7. Extreme Compression and Cirrhosis of the Lung. A Preliminary Experimental Study.
   Dr. C. M. Van Allen, Peiping, China.

8:00 p.m. PRESIDENT'S BANQUET.

Thursday, May 11

9:00 a.m.

1. Bronchiectasis.
   Dr. James A. Miller, New York City.
2. Selective Surgical Collapse for Lung Abscess.
   Dr. Richard H. Overholt, Boston.
3. Fixation of Chest Lesions With Subsequent Compression.
   Dr. W. P. Herbert, Asheville.
4. Oidiomycosis of the Lungs.
   Dr. David T. Smith, Durham, N. C.
5. Experimental Pulmonary Lesions of Aspergillus Niger: Superimposition of This Fungus on Experimental Pulmonary Tuberculosis.
   Dr. Norman Bethune, Montreal.
6. Suture of the Lung.
   Dr. Frank S. Dolley, Los Angeles.
7. Intrapleural Pneumolysis. A Critical Review.
   Dr. Julian A. Moore, Asheville.